VOLUME 8 *Chicago Number* NUMBER 2

THE MEDICAL CLINICS OF NORTH AMERICA

SEPTEMBER, 1924

PHILADELPHIA AND LONDON

W. B. SAUNDERS COMPANY

PUBLISHED BI-MONTHLY (SIX NUMBERS A YEAR), BY W. B. SAUNDERS COMPANY, WEST WASHINGTON SQUARE, PHILADELPHIA

MADE IN U. S. A.

CONTRIBUTORS TO THIS NUMBER

ISAAC A. ABT., M. D., Professor of Diseases of Children, Northwestern University Medical School; Attending Physician, Diseases of Children, Sarah Morris Children's Hospital of Michael Reese Hospital.

WILLIAM A. BRAMS, M. D., Adjunct Attending Physician, Michael Reese Hospital; Assistant Professor of Internal Medicine, Post-Graduate Hospital and Medical School.

ARTHUR F. BYFIELD, M. D., Assistant Professor of Medicine, Rush Medical College; Attending Physician, Cook County Hospital.

JAMES G. CARR, M. D., Associate Professor of Medicine, Northwestern University Medical School; Attending Physician, Cook County, Wesley Memorial, and South Shore Hospitals.

EDWARD LYMAN CORNELL, M. D., Associate in Obstetrics, Northwestern University Medical School; Attending Obstetrician, Chicago Lying-in, Auburn Park, and Illinois Masonic Hospitals.

ARTHUR R. ELLIOTT, M. D., Professor of Medicine, University of Illinois College of Medicine; Physician to St. Luke's Hospital.

JOSEPH C. FRIEDMAN, M. D., Attending Physician, Michael Reese Hospital.

JESSE R. GERSTLEY, M. D., Associate in Pediatrics, Northwestern University Medical School.

CLIFFORD G. GRULEE, M. D., Clinical Professor of Pediatrics, Rush Medical College (in Affiliation with the University of Chicago); Chief of Pediatric Staff, Presbyterian Hospital.

WALTER W. HAMBURGER, M. D., Assistant Clinical Professor of Medicine, Rush Medical College; Attending Physician, Cook County Hospital; Associate Attending Physician, Michael Reese Hospital.

RALPH C. HAMILL, M. D., Assistant Professor of Neurology, Northwestern University Medical School; Attending Neurologist, Wesley Memorial Hospital; Consulting Neurologist, United States Public Health Service, Chicago.

JULIUS H. HESS, M. D., Professor and Head of the Division of Pediatrics, University of Illinois College of Medicine; Chief of Pediatric Staff, Cook County Hospital; Attending Pediatrician to Cook County, Michael Reese, and Englewood Hospitals; Consulting Pediatrician, Municipal Contagious Hospital, Chicago; Member of the Advisory Committee to the Children's Bureau, Department of Labor, Washington, D. C.

LAWRENCE E. HINES, M. D., Instructor in Medicine, Northwestern University Medical School; Attending Staff, St. Joseph's and Chicago Memorial Hospitals.

CHARLES LOUIS MIX, M. D., Professor of Medicine, Loyola University Medical School.

JACOB MEYER, M. D., Adjunct Attending Physician, Michael Reese Hospital; Associate Attending Physician, Mt. Sinai Hospital; Instructor in Medicine, University of Illinois College of Medicine.

MILTON M. PORTIS, M. D., Chicago.

ROBERT SONNENSCHEIN, M. D., Professor of Otolaryngology, Post-Graduate Medical School; Assistant Professor of Otolaryngology, Rush Medical College of the University of Chicago; Consulting Otolaryngologist, Durand Hospital of the McCormick Institute for Infectious Diseases; Associate Attending Otolaryngologist, Michael Reese Hospital.

IRVING F. STEIN, M. D., Associate Attending Obstetrician and Gynecologist, Michael Reese Hospital; Associate in Obstetrics, Northwestern University Medical School.

L. J. WILHELMI, M. D., Resident Physician, Sarah Morris Hospital for Children.

CHARLES SPENCER WILLIAMSON, M. D., Professor of Medicine and Head of the Department, College of Medicine, University of Illinois; Attending Physician, Cook County Hospital.

CONTENTS

Clinic of Dr. Isaac A. Abt, *Michael Reese Sarah Morris Hospital for Children*
A Case of Aleukemic Leukemia with Clinical Symptoms of Plastic Anemia..... 427

Clinic of Dr. Charles Spencer Williamson, *Cook County Hospital*
Three Cases of Tuberculous Peritonitis..... 437
Primary Carcinoma of the Liver..... 453

Clinic of Dr. Joseph C. Friedman, *Michael Reese Hospital*
The Interpretation of Prolonged Minimal Temperatures..... 465
A Case of Pneumolithiasis..... 477

Clinic of Dr. Ralph C. Hamill, *Northwestern University Medical School*
Tics..... 483

Clinic of Dr. Arthur R. Elliott, *St. Luke's Hospital*
Cardiac Aneurysm..... 495

Clinic of Dr. Jacob Meyer, *Michael Reese Hospital*
Subphrenic Abscess—Suppurative Pylephlebitis Secondary to a Perforating Gastric Ulcer..... 519

Clinic of Dr. Irving F. Stein, *Michael Reese Hospital*
Sterility..... 525

Clinic of Dr. William A. Brams, *Michael Reese Hospital*
Plaque-like Adenoma of the Stomach, Its Differential Diagnosis and Treatment 533

Clinic of Dr. Robert Sonnenschein, *Post-Graduate Hospital*
A Series of Unusual Ear, Nose, and Throat Cases..... 539

Clinic of Dr. Lawrence E. Hines, *Northwestern University Medical School and St. Joseph's Hospital*
Cardiovascular Syphilis..... 559

Clinic of Dr. Julius H. Hess, *Cook County Hospital*
Diaphragmatic Hernia (Eventration, True and False Hernia) In Infants and Children..... 567

Clinic of Drs. Jesse R. Gerstley and L. J. Wilhelmi, *Northwestern University Medical School*
Non-operative Treatment of Pyloric Stenosis in Infancy..... 587

Clinic of Dr. Charles Louis Mix, *Mercy Hospital*
Chronic Nephritis..... 603
Cardiospasm..... 619
Mucous Colitis..... 627

Clinic of Dr. Walter W. Hamburger, *Michael Reese Hospital*
The Prevention of Heart Disease..... 631

Clinic of Dr. Milton M. Portis, *St. Luke's Hospital*
Multilocular Cyst of the Pancreas..... 641
Surgical Indications in Peptic Ulcer..... 647

Clinic of Dr. Clifford G. Grulee, *Presbyterian Hospital*
Acute Hemorrhagic Nephritis in Children..... 653

Clinic of Dr. James G. Carr, *Cook County Hospital*
Secondary Hypertrophic Osteo-arthropathy..... 663

Clinic of Dr. Arthur F. Byfield, *Cook County Hospital*
The Diagnosis of Splenomegaly with Hematemesis..... 681

Clinic of Dr. Edward Lyman Cornell, *Chicago Lying-in Hospital*
Pyelitis Complicating Pregnancy..... 693

THE MEDICAL CLINICS
OF
NORTH AMERICA

Volume 8 Number 2

CLINIC OF DR. ISAAC A. ABT

MICHAEL REESE SARAH MORRIS HOSPITAL FOR CHILDREN

A CASE OF ALEUKEMIC LEUKEMIA WITH CLINICAL SYMPTOMS OF PLASTIC ANEMIA

THE patient that I showed you in a preliminary way several weeks ago, who had a marked anemia and leukopenia with swelling and tenderness over the right tibia, died during the past week. The patient came to autopsy. I wish to review with you the history, and ask you to listen to the autopsy report, and to consider in detail the clinical course of the disease and the diagnosis as revealed by the postmortem examination.

You will remember that when I presented her we made a provisional diagnosis of "aplastic anemia." This little girl, S. C., aged five years, was admitted to the hospital complaining of pain over the right tibia which had been present for the past two weeks. One year ago she fell and hurt her shin. Following this she had a swelling in this region, which persisted for some time and recurred at intervals.

Abstract of History.—Our little patient, Sylvia, was born at full term, and her first period of life was uneventful. The baby's mother had pneumonia during pregnancy, though the infant developed normally. She sat up at six months, walked at ten months, talked at twelve months. She was breast fed until she was eleven months old. Her first tooth erupted at ten months. Thus it is observed that her progress during the first year was in every respect satisfactory except that she had whooping-cough at eight months. She had the usual infectious

diseases—measles at one year, chickenpox at four years, and mumps at five years.

Clinical Description.—As has already been noted, the patient had a swelling on the crest of her tibia which appeared about two months ago, and disappeared two weeks before admission to the hospital. On entrance she complained of great pain and tenderness over the right tibia. This pain was sharp and severe, though the patient said it was not constant. It was intermittent and usually lasted from one to five minutes. The pain aroused her from sleep. The greatest point of tenderness was over the crest of the right tibia in the upper third. The inflamed area was about 2 inches in length. The region was red and swollen. She had a temperature of 100° F. on admission, which rose to about 101° F. on the fifth day. On the eighth day it reached 103° F., showing a morning decline. Shortly before death the temperature rose to 104° F.

The admitting physician referred the child to the surgical service because he suspected an acute bone disease. She was promptly transferred to the medical department.

The principal observations showed that the tonsils were considerably enlarged, though not inflamed. There were some small palpable lymph-nodes occurring in chains in the anterior and postcervical regions and also in the supraclavicular regions. The glands were the size of shot, soft and discrete. The inguinal and axillary glands could be palpated. The epitrochlears could not be felt. There was a short systolic murmur over the heart which was not transmitted. The liver could be felt one and one-half fingerbreadths below the costal arch. The tip of the spleen could be felt only on deep inspiration. The skin and mucous membranes were pale.

About two weeks after admission she developed some pain and tenderness over the left wrist-joint, and some limitation of motion resulted. This lasted only a few days, and disappeared. Shortly after this she complained of preauricular pain, though there was no tenderness or swelling. She had now been in the hospital for more than two weeks. The heart-rate was rapid, and the murmur over the mitral area had increased in intensity,

though it was not transmitted into the axilla. The spleen remained palpable, though it was not markedly enlarged. About this time she had a nosebleed from the left nostril.

Four days before death the temperature rose to 104° F., after a blood transfusion. Toward the end she developed a few scattered râles over the right chest posteriorly. The liver increased somewhat in size. The spleen remained barely palpable. An eruption appeared over thighs and legs which consisted of papules about the size of rice kernels. Some of these became vesicular. Later in the day the same eruption appeared on the arms and trunk and palms of the hands. It was also observed that those which appeared first on the lower extremities became purpuric in character. Finally, there was a generalized purpura.

The various blood examinations are all characterized by a marked reduction in hemoglobin and number of red blood-cells, and by persistent leukopenia and a marked preponderance of small lymphocytes. Urinary examinations were normal. Stools showed no abnormalities. The Pirquet and Wassermann tests were negative. The *x*-ray examination of the right tibia showed a definite periosteal thickening in the upper third.

The patient expired after having been in the hospital twenty-one days. The autopsy was performed by Dr Oscar T. Schultz, assisted by Drs. B. Portis and W. Blum.

The most important points elicited consisted of the following findings: The liver extended three fingerbreadths below the costal arch, was enlarged, and very firm. The lobular markings were indistinct, and fatty changes were noted The thymus was converted almost entirely into a mass of fat. The blood was dark and unusually watery. There was nothing of any importance in the examination of the heart and lungs. The spleen was moderately enlarged. It was very dark and firm. The capsule was wrinkled. The cut surface showed a considerable increase of fibrous tissue. The mesenteric lymph-nodes were all noticeably enlarged and the periaortic lymph-nodes were enlarged. The pelves of the kidneys were studded with small petechial hemorrhages. The mucosa of the bladder also contained some hemorrhagic foci.

The tibia of the right side in its upper portion showed an absence of inflammation or necrosis of bone or subperiosteal hemorrhages. The fresh section of the bone revealed almost an entire fat replacement of the marrow, so that it assumed a yellowish color. There was a dense, compact layer, about 3 mm. in thickness, which completely encircled the marrow cavity. This layer was likewise encircled by a dense, compact, bony layer measuring about 2 mm. in thickness. Finally, there was a thickened layer of periosteum over the outermost osseous structure.

Microscopic Examination.—Liver and kidney sections showed a definite infiltration, with fairly large round-cells, with light staining nuclei. Some of these were eosinophils. The impression was that this cellular infiltration was not inflammatory, but rather an infiltration of young blood-cells. In the kidneys the same condition was observed, namely, the periglomerular infiltration with round-cells, and this infiltration also was observed about some of the smaller blood-vessels of the kidney. The lymph-nodes of the mesentery and ovary showed similar pathologic changes, namely, the round-cells were clumped about the blood-vessels. Section through a rib showed that the line of ossification was regular. The marrow was active. It did not seem hyperplastic.

Section Through the Affected Part of the Tibia.—The medulla was filled with closely packed round-cells which varied in size and in chromatin content, and which presented numerous necrotic areas. The bone trabeculæ were missing from the medulla. There was a thick layer of very compact bone, and external to this there was a radial proliferation of bone. The interstices were filled with young fibrous tissue which contained quite a few round-cells which did not stain well. This unusual bone formation was covered with rather firm fibrous tissue and a thin layer of round-cells.

This case is interesting and instructive because it illustrates the difficulty in the exact diagnosis of some of the blood disorders which we encounter during infancy and young childhood. The progressive anemia, the marked reduction in erythrocytes, the

persistent leukopenia, and the preponderance of small lymphocytes led us to assume that the condition was an aplastic type of anemia. The patient at no time while under our observation showed any tendency toward regeneration of erythrocytes. The spleen was only barely palpable and the lymph-node involvement was not significant.

Baar[1] points out that 41 cases of aplastic anemia were collected, of which 5 were in children. A positive diagnosis in these could not be verified. One must be very guarded in making a diagnosis of aplastic anemia in children, since it is much less frequent than acute leukemia. Baar suggests that one is justified in speaking of an aplastic anemic blood-picture in an acute leukemia, but not of a combination of the two.

Referring back to our own patient, we are reminded that the child showed an aplastic state on the basis of a leukemic process. It is true that localized bone involvement, such as occurred in the upper third of the right tibia, is not characteristic for aplastic anemia. On the other hand, periosteal thickening or tumor formation, such as occurs in chloroma, is rarely found in typical cases of lymphatic leukemia. Domarus tells us that in cases of aleukemic leukemia bone changes are rarely observed. Sometimes there is a periosteal proliferation associated with infiltration of leukemic tissue.

I think it will be admitted that the blood findings in our little patient showed a lack of regenerative power of the blood elements—an aplasia. Whatever may have been the cause, it has already been pointed out that the pathologist's report showed that there was a leukemic infiltration into various organs. This compels us to accept the diagnosis of aleukemic leukemia. This condition is a true leukemia without leukocytosis of the blood. There are certain cases of leukemia in which the leukocyte count may be normal during some stage of the disease, and others in which there probably never is a leukocytosis.

Aleukemic leukemia has also been designated as "pseudoleukemia." The use of this term should be avoided because it confuses this condition with Hodgkin's disease. Aleukemic

[1] Jahrb. f. Kinderheilk., 1924.

leukemia may be either of the lymphatic or medullary types. This stage may precede the actual occurrence of a typical leukemia, or the blood types which represent failure of regeneration may occur as a period of remission during an active leukemic process.

Domarus[1] describes the varieties and the differential diagnosis of aleukemic leukemia:

In the symptom-complex of aleukemic lymphadenosis the localization of leukemic hypertrophic processes is especially in the lymphatic organs, the lymph-glands, and spleen. Other organs may also be affected, the lymphatic tissue contained in them undergoing hyperplasia. The proliferating tissue shows a definite tendency to infiltrate the surrounding tissue like a malignant tumor and to form large tumor masses. After the various cases are compared the aleukemic proliferation can be classified into definite groups with their individual characteristics:

Chronic Forms.—(1) The group which is most frequent and most easily recognized clinically is the form in which lymphomata form on the neck, in the axilla, in the groin, and in the body cavities. There is also a moderately enlarged spleen and a relative increase of lymphocytes in the blood. There is a regularity in the distribution of lymphocytes over the entire body. (2) In rare cases the gland swelling may be secondary; and the splenic tumor dominates the disease picture. This form has been designated as *splenic pseudoleukemia*. This may offer diagnostic difficulty, especially if lymph-glands are not greatly enlarged. (3) In other forms of leukemia the tumor-like characteristic of leukemic tissue predominates. To this group belong the forms which show sarcoma-like proliferation, originating from thymus and the glands of the mediastinum, and in which the symptom-complex of mediastinal tumor appears. The enlarged lymph-nodes often fuse and, therefore, behave like a true malignant new growth. In a small portion of the cases there are other tumor-like proliferations besides the mediastinal tumor, for example, infiltrated growth in the pharynx and in the larynx. Infiltration in the orbit and the eyelids have also been reported.

[1] Kraus and Brugsch, Spez. Path. u. Ther., 1917, v. viii, p. 478 ff.

The lymph-glands in the neighborhood enlarge, and clinically this picture resembles lymphosarcoma.

(4) Another characteristic group resembles Mikulicz's disease—chronic enlargement of the lacrimal and salivary glands due to the replacement of glandular tissue by lymph-cells. (5) The localizaton of the leukemia in the digestive tract is rare. It is characterized by gastro-intestinal symptoms with diarrhea, and there may be symptoms of intestinal obstruction. (6) There are rare cases of aleukemia which are characterized for a certain period by the clinical picture of a severe anemia with or without lymphocytosis without lymphoma or other signs of a lymphatic hyperplasia. Possibly slight enlargement of the spleen may point to the true character, especially when there is a marked lymphocytosis in the blood. Cases of this kind, which are very rare, have been called medullary pseudoleukemia, which signifies that the localization of the aleukemia is exclusively or predominatingly in the bone-marrow, whereas the other organs which usually participate in the disease, especially the lymph-glands and spleen, are either slightly or not at all affected.

Litten, Runeberg, and v. Baumgarten were the first to describe cases of this kind (lymphadenia ossium, myelogenous pseudoleukemia). In a case of Senator, a thirteen-year-old boy showed a progressive anemia, with normal number of leukocytes, with relative lymphocytosis of 83 per cent. Neither gland swellings nor enlargement of the spleen were present. On autopsy a diffuse lymphoid change of the bone-marrow was found.

In the observation of Rubinstein a lymphocytosis of 68 per cent., with an extreme leukemia, was found. Domarus reports a boy of six years, sick for some time, with blood finding of 3.2 million erythrocytes and 55 per cent. hemoglobin, and diminution of leukocytes to 3200, with 75 per cent. small lymphocytes and 13 per cent. large lymphocytes. There was no enlargement of the spleen, liver, or lymph-glands. A few small palpable glands were present in the neck. Diagnosis of aplastic anemia was made, since the characteristic signs for regeneration of erythropoiesis were absent. One year later he died. On autopsy, marked enlargement of the spleen and liver, swelling of a few

lymph-glands, and red marrow were found. Microscopically, the marrow was found in diffuse lymphoid metaplasia; the spleen showed the picture of a chronic lymphatic leukemia, with disappearance of its structure. In the liver there were typical lymphomata, as in leukemia. The histologic examination of the lymph-glands was normal for the most part. Some of the lymph-sinuses were very cellular. In the tonsils marked lymphatic hyperplasia with infiltration of the surrounding musculature was found. Smears of the blood, postmortem, showed a picture of a lymphatic leukemia with large lymphocytes predominating. This case is a characteristic example of the aregenerative stage of leukemic affection, so that the first diagnosis in the case reported was aplastic anemia.

Undoubtedly the disease originated in the bone-marrow. The diffusion to other organs indicates a leukemic proliferation. This was, therefore, a pure medullary localization of the leukoblastic hyperplasia which at first took an aleukemic course and later became leukemic. This case indicates that the leukemic and pseudoleukemic disease forms are different symptoms of one and the same anatomic process.

It is pointed out that the diagnosis of medullary pseudoleukemia should be made only with the greatest caution *intra vitam*. There are numerous severe anemias without lymph-gland or spleen enlargement in which there is a relative lymphocytosis, but which turn out to be small myeloblasts instead of lymphocytes. The diagnosis of the medullary form of leukemia can be made with certainty only on histologic analysis.

Diagnosis and differential diagnosis: For the first group, in the presence of multiple lymphomata, splenic tumor, and relative lymphocytosis, the diagnosis is clear.

Etiology.—Baar[1] states that a satisfactory explanation of the cause of aleukemic processes has not yet been found. He summarizes the various opinions concerning the failure of blood-cells to regenerate in aleukemic leukemia.

Pappenheim states that the condition for the establishment

[1] H. Baar, Acute Aleukocythemic Leukemia in Childhood, Jahrb. f. Kinderheilk., 1924, civ, 1–32.

of a leukemic blood-picture is definite disease of the bone-marrow. Neumann's theory of the development of leukemia is the following: If the pathologic growth stimulus involves primarily or exclusively the spleen or lymph-glands whose elastic capsule grows, then only pseudoleukemia results; if the bone-marrow is affected in the same way and becomes hyperplastic, leukemia results. These assumptions are false, since true leukemia without affection of the bone-marrow and, on the other hand, pseudoleukemia with affection of the bone-marrow, have been described.

Herz interprets a case of leukopenia by saying that there is an extremely deleterious effect of an infection on the blood-forming organs which lead to the complete disappearance of the granulocytic system. Later, perhaps after a certain adaptation to the organism, excessive new formation and hyperplasia may result, the most immature type of cells being formed which were not discharged into the blood-stream, perhaps because the patient did not live long enough. This explanation cannot hold for all aleukocythemic leukemias, at least not for the cases with leukopenia of any duration.

Zypkin tries to find the basis for the difference between pseudoleukemia and leukemia in the biologic behavior of the cells. He distinguishes four types of aleukemic leukemia: The first type includes the cases in which the leukocytic blood composition is approximately unchanged quantitatively and qualitatively. The second type includes the cases with leukopenia without essential changes in the qualitative composition of leukocytes. The third type comprises the cases with leukopenia and change of the qualitative composition of the white corpuscles, either in the sense of a lymphemia or myelemia. The fourth type is composed of the cases which show normal or increased number of leukocytes, while the white blood-picture is changed in the sense of a lymphemia or myelemia. The four types of aleukemic leukemia are believed to correspond to differences in severity of disturbance of normal leukopoiesis caused by a hyperplastic process which differs only in degree. The occurrence of leukemic blood composition is believed to be due to a progressive increase in the strength of the toxin which leads to such marked prolifera-

tion that the products of proliferation no longer find room in the blood-forming organs and, therefore, begin to overwhelm the peripheral blood. The cells are believed to proliferate the more, the lower their order and the more embryonal they are. The assumption that the blood-forming organs in leukemia are composed of cells which are more embryonal than in aleukemic leukemia does not find support in the histologic findings. Thus, in acute aleukemic leukemia the bone-marrow often consists of myeloblasts, which are very young cells.

In cases of acute leukemia it may be assumed that an injurious agent, perhaps the same which leads to the proliferation of the myeloid or lymphatic tissues, acts as a chemotactic stimulus to the blood-making organs. But wherever there is positive there is also negative chemotaxis. In this way the occurrence of leukemias with leukocytic increase in the blood or with leukemia may be explained. This assumption does not seem unfounded when it is considered that for most normal and pathologic blood-cells active motility has been demonstrated. This has been known for a long time so far as the polymorphonuclear neutrophils and the large mononuclears are concerned. It has been proved by Wolff and Bergel for the lymphocytes. Erb observed phagocytosis in lymphoid cells in acute leukemia. Jakobsthal saw phagocytosis in myeloblasts.

From the data which I have submitted to you you will observe that this little patient presented the symptoms of an aplastic anemia, though with the histologic findings of an aleukemic leukemia. Possibly the main emphasis should be laid on the fact that the blood diseases of childhood may present difficulties in clinical diagnosis.

The outstanding point is that the lymphatic leukemic type is relatively frequent and that it may present itself in various forms, and that the blood examination may be misleading in that the number of leukocytes and erythrocytes may be markedly diminished. In the cases, however, where there is a marked lymphocytosis, even in the presence of leukopenia, a lymphatic leukemia should be suspected.

CLINIC OF DR. CHARLES SPENCER WILLIAMSON

COOK COUNTY HOSPITAL

THREE CASES OF TUBERCULOUS PERITONITIS

CASE I. TUBERCULOUS PERITONITIS. CASEOUS (ULCERATIVE) TYPE

THE first case which I wish to present today is that of a colored man, fifty years of age, who came into the hospital between five and six weeks ago, complaining of the following conditions:

Present Complaint.—The most striking thing noted was pain in the abdomen, which he said had existed more or less continuously for six months. It was a dull, gnawing pain, never of great severity, almost constantly present, and which he describes as "drawing across the upper portion of the abdomen." Occasionally it will assume a sharper character until it becomes quite excruciating. This he notices happens mostly at night. These excruciating exacerbations last only a short time, tapering off again into the normally dull pain.

Vomiting.—He vomited during the week before his entrance into the hospital, the vomited material being only the food taken. This has occurred after every meal, but he has never noticed any bile or blood present in the vomitus. On direct questioning he states that he had vomited for some time previous to admission, but at rather long intervals, and he was inclined to think that it was as a result of poor food which he had eaten.

Constipation.—This symptom existed for several months before admission, so that he had to take medicine for the bowels almost every day. During the week before admission he suddenly developed a diarrhea which consisted of loose movements, "like water," and averaged from three to five times a day.

Further questioning elicits the fact that he had had a number of attacks of diarrhea lasting for a day or two during the six months before admission, but had paid little attention to it.

Loss of Weight.—There has been a fairly marked loss of weight. A year before admission he weighed 125 pounds, and he has noted from the way his clothes hang upon him that he has lost a moderate amount of weight. For the five or six weeks before entering this hospital he had been in bed at home because of his extreme weakness. He hardly knows when the weakness became so severe as to cause him to cease working, but insists that it came on rather gradually.

When we inquire as to other *gastro-intestinal symptoms* we find that his appetite had been pretty good until a week or two before admission. The vomiting, constipation, and diarrhea have already been described, and he is quite sure that he has had no blood in the stools at any time, stating that he has paid special attention to this.

Genito-urinary Symptoms.—At times he has had pain on urination at a point located just behind the scrotum, and being especially marked at the end of urination. He has never noticed anything abnormal about the urine. He has considerable frequency during the day and has had to empty the bladder from two to six times every night during the past three months.

His past *medical* and *surgical history* shows nothing which casts any light on the present condition. He had gonorrhea twice when young. He denies syphilis. I may say that his blood Wassermann has been negative.

Family History.—He knows very little about his family except that he has lost 4 children of fever in early childhood.

Habits.—He neither smokes nor drinks and is not a drug user.

If we attempt to analyze this patient's condition on the basis of the symptoms you have just heard, we could hardly come to any very definite conclusion, except that there seems to be some condition associated with the gastro-intestinal tract.

Physical Examination.—Proceeding now to the physical examination of the patient, as described in the history at the time

of admission, we see that he was afebrile on admission, temperature 96.4° F., pulse 120, and respirations 20.

Eyes, ears, head, and *neck* were entirely negative except for a large number of very bad teeth.

Chest.—Careful examination of the lungs revealed nothing abnormal and the *x*-ray showed nothing pathologic.

Heart.—The apex impulse has the normal position, there is no enlargement of either ventricle; no murmurs present. Blood-pressure 130/80.

Abdomen.—This seems slightly distended. It is tympanitic everywhere, and the most careful examination failed to reveal any evidences of fluid. The liver and spleen were of normal size and the kidneys were not palpable. There was some tenderness throughout on deep pressure, which was slightly more marked in the epigastrium.

Genitalia.—Negative. The inguinal glands and those along the femoral vessels are somewhat enlarged.

Extremities.—These show nothing abnormal. The deep reflexes are all O. K.

Rectal examination shows external hemorrhoids, and a moderately enlarged and smooth, but not tender, prostate could be palpated.

If we attempt to build a diagnosis upon the results of the physical examination we shall have to say that all that has been found was a somewhat distended, tender abdomen, with no evidences of ascites. The patient is moderately emaciated and apparently quite weak. Because of this we had a careful blood examination made which revealed red corpuscles, 2,300,000; whites, 13, 600; hemoglobin, 42 per cent. The differential count showed 85 per cent. polymorphonuclear neutrophils, 11 per cent. small lymphocytes, 3 per cent. large lymphocytes, and 1 per cent. transitionals. There was a moderate degree of poikilocytosis. Aside from the severe anemia which is manifestly secondary, we do not learn much from the blood examination. The urinalysis was negative in every respect. Analysis of the stomach contents and stools likewise failed to shed any light upon the condition.

While the patient was afebrile at the time of his actual admission to the hospital, on subsequent days he was seen to have a little fever, running from 99.2° to 100° F. by rectum, and as the weeks have gone by his fever has been maintained at about the same height, rarely going over 101° F. and semi-occasionally coming down to a degree or two below normal.

Now, what are the possibilities in a case of this kind? Among the first things to be thought of, although by no means the most common, would be some of the less common intestinal parasites. I have recently seen several cases of this sort due to various types of flagellates. While it is not always easy to find such flagellates at every stool examination, yet in this case repeated search has failed to show them, so that I think we are quite safe in concluding that this is not the underlying pathology. It may not be amiss to give you a word of warning in regard to drawing too many conclusions from the finding of a few flagellates. This has led to a good deal of misconception as to their importance, and it is only when they are present in enormous numbers and definitely associated with diarrheal attacks should they be regarded as a causative factor. Aside from these, the two things which are most likely to be thought of are pernicious anemia and tuberculosis. The results of the blood examination alone are quite sufficient to rule out the former, and so our thoughts are directed to a consideration of tuberculosis either of the bowels or of the peritoneum. At first blush, one might think that the alternating diarrhea and constipation would suggest tubercular enteritis rather than a tuberculosis of the peritoneum. The stools were carefully examined for tubercle bacilli by the more recent methods, but none were found. Now let us consider how the case stands with regard to tuberculous peritonitis. Does it act in this way? In one group of cases the symptoms are almost exactly parallel to those which are presented by this patient. I refer to the chronic type without much exudation and without much fibrosis. The symptoms in a patient with this condition generally point to the abdomen, but by no means as definitely as you might suppose from the extensive nature of the pathology which is found after death. The clinical history

begins with irregular colicky pains, often with dull pain in between, and quite commonly intensified by movements such as bending and twisting, which bring pressure on the abdominal contents. Not infrequently both urination and defecation are attended with some pain and frequency of urination is very common. The bowels are generally constipated and yet attacks of sharp diarrhea are constantly found. There is progressive loss of weight, a progressive secondary anemia with frequently a slight increase in the leukocytes, and a very little fever. Occasionally there is a slight leukopenia. Sometimes the cases are almost afebrile. Tenderness over the abdomen is quite common and may have its point of maximum severity almost anywhere, generally around the navel.

Comparing the symptoms I have just described with those presented by this patient, you can see that this is, in reality, a text-book case. In the weeks which he has spent in the hospital under our observation the condition has apparently remained almost unchanged, except that each week seemed to find him a little more apathetic and weak. Some time ago we had a careful *x*-ray study of the bowel made, which showed nothing positve except that the cecum at a point just above its head was firmly fixed. It was noted too that this part of the abdomen was becoming increasingly tender.

Several special methods of diagnosis were attempted. We managed to secure a little sputum, but could find no tubercle bacilli. I do not consider it wise to attempt a tuberculin test in a patient of this sort, as it is dangerous and rarely ever leads to any real decisive information. One could, of course, do a von Pirquet, and might draw some conclusions if this were negative, but we did not do so. Proctoscopic examination showed nothing especial except considerable pain when the tube was inserted to its full length, the pain apparently being caused by the traction on the bowel.

Our diagnosis, then, which was made definitely about a week after entrance to the hospital, was tuberculous peritonitis with caseous glands throughout the abdomen.

I show you this case as a classical example of one of the types

of tuberculous peritonitis, namely, the type which is chronic in character, running its course without ascites, frequently, perhaps generally, associated with extensive caseation of the glands in the abdominal cavity, and which is very often associated with tuberculosis of the contiguous viscera, such as a tuberculous enteritis or a tuberculosis of the cecum.

Note.—Since this case was presented the patient died suddenly and rather unexpectedly. A postmortem was made and a summary of the findings is as follows:

Anatomic Diagnosis.—Hyperplastic and caseous tuberculosis of the mesenteric lymph-glands; diffuse fibrinocaseous tuberculous peritonitis; nodular caseous tuberculosis of the spleem.

The interesting feature is that the lungs and tracheobronchial glands were quite negative, except for one or two very old fibrous patches which could not certainly be stated to be of a tuberculous nature.

CASE II. TUBERCULOUS PERITONITIS (FIBROID TYPE)

The second patient is again a colored male, aged twenty-five, who entered the hospital a week ago with two chief complaints, pain in the upper abdomen and back and weakness. These symptoms have lasted three months. The onset was gradual, and along with this pain he developed an indigestion, with nausea and much belching, but no vomiting. These symptoms gradually grew worse, and two or three weeks before admission the patient developed considerable pain and soreness over the abdomen. At times he had nausea, which never lasted more than a day or two, and, although he tried to vomit, he could not. After awhile the pain left the rest of the abdomen and, as he expressed it, "drew around to the left chest," where it has stayed and where it exists at the present time. He says he has always had a weak back, but that the pain there is different from the pain in the back of which he now complains.

Respiratory Symptoms.—He has no cough, no shortness of breath, no hoarseness, and no pain except that described in the side.

Gastro-intestinal.—Appetite is generally fair and he has

no difficulty in swallowing. The pain which began in the left upper abdomen is much better and is now only a sense of soreness. There is much gaseous distention, which comes on especially at night, and if he is constipated this symptom is greatly exaggerated. In his judgment the gas is the cause of most of his trouble. He states that six or eight weeks ago he had some swelling in the abdomen, which after a short time subsided, with a great deal of belching and flatus. Bowels are regular and he has not had any attacks of diarrhea and has not noticed any blood in the stools. There is no distress on defecation.

Cardiovascular Symptoms.—Except for a little dizziness and faint feeling during the last few weeks these are entirely absent.

Genito-urinary.—He volunteers the statement that he has had to urinate much more frequently in the past week or two, and at night this is so urgent as to cause him a great deal of trouble, and he has frequently had to get up six or seven times during the night. He experiences no difficulty in passing urine, the stream being free and forceful and without burning. Parenthetically his prostate is quite normal and the urine examination is entirely negative.

The patient himself lays much stress on his lumbar pain. He says it is a dull, grinding pain, worse at night on lying down, and not very much influenced by bending over.

Nervous symptoms are absent.

There has been considerable loss of weight—about 15 pounds in the last five months—and the weakness has been steadily progressive.

Previous and pathologic histories disclose nothing of importance. He has had gonorrhea and one attack of malaria.

By occupation he is a stock-yards laborer and drinks no alcoholics.

I think it would be advantageous to look over the patient before discussing the diagnosis, and I may say that his physical condition is just about what it was when he first entered the hospital.

Physical Examination.—Head and neck negative except for a slight enlargement of the anterior cervical glands.

Lungs show a few crackles over the right upper lobe, both anteriorly and posteriorly. These have been present at each examination and have given rise to the probable diagnosis of a chronic pulmonary tuberculosis, but we have not been able to find any bacilli in the sputum, although these have been looked for daily.

Heart is negative.

Abdomen.—Getting the patient in a good light you can see that the abdomen is rather more rounded than you would expect in a thin individual, and is firm and somewhat doughy. It is quite tympanitic all over and there is no dulness in the flanks. On placing the patient in the modified knee-chest position with the trunk horizontal, we find no area of flatness around the navel. I recommend this position to you as being the position in which one can most readily determine minimal quantities of fluid. The liver and spleen cannot be felt, but above the navel and extending to either side one can feel quite plainly the masses the size of a small fist, which are rather fixed, slightly tender, especially on the left side. There are no abnormal pulsations and no enlargement of the subcutaneous veins. The kidneys are not palpable. The extremities show nothing abnormal. The temperature on admission was 98.4° F., and since then he has been afebrile most of the time, once or twice the temperature rising to 100° F. per rectum. Blood-pressure is 102/78; pulse, 70; respirations, 24.

The blood examination reveals, the first time, red blood-corpuscles 4,000,000, whites 8400, hemoglobin 50; the second time (yesterday), 4400 whites, a differential count of 52 per cent. polymorphonuclears.

A complete gastro-intestinal *x*-ray examination was made, with practically normal findings. The rectal examination was negative. A test-meal was given and was found to be normal. The stools were examined, both for blood and parasites, all with negative results.

Diagnosis.—You can hardly fail to have observed the great similarity of this case with the first one presented, and yet there are distinct differences to which I wish to call attention. Both

start in with weakness and rather indefinite abdominal symptoms. The first case showed occasional attacks of severe diarrhea; this one is constipated all the time, but at times he "balloons up" and belches a great deal of gas and passes it by rectum, after which he is more comfortable. The first patient showed very little fever, and this one even less. Neither case has shown any ascites, but the patient now before you shows definite, doughy, rather firm masses in the abdominal cavity, slightly tender, and the impression given by the abdomen as a whole is that it is larger than normal, the distention being due to distended bowels. The case before you might be thought to be one of carcinoma, although the age of the patient (twenty-five) would speak against this. We must, however, remember that, particularly in the gastro-intestinal tract, carcinomata are not infrequent at this age or even younger. I have myself seen several instances of carcinoma of the large bowel in patients in the twenties. However, we have the very important negative testimony of the *x*-ray; second, the symptoms are not those usually found with carcinoma of the bowel; third, while there is very little fever, yet there is some, and this is usually not the case with carcinoma. and last, and most important of all, we have slight but quite definite signs in the lungs pointing toward a pulmonary tuberculosis. So we have no hesitancy in stating that this is another case of tuberculous peritonitis of a still different type. The patient before you is a typical example of a chronic fibrous tuberculous peritonitis, the fibrosis having gone on to such an extent as to cause a great thickening and puckering of the omentum and a matting together of the intestines into these very characteristic doughy, moderately firm, and somewhat tender masses. To the uninitiated these would almost certainly be taken for genuine neoplasms.

This particular type of tuberculous peritonitis differs from the first in its chronicity, which is very great. While he describes his symptoms as having lasted only a few weeks or months, I think we can be quite certain that they have lasted very much longer, since such a degree of fibrous formation requires a long time for its development. In view of the fact that we have

quite certainly an old fibrous tuberculosis in the lung, although of no great extent, it is altogether probable that we will find some of the abdominal viscera, possibly the bowel itself, and almost certainly the mesenteric glands, involved in the tuberculous process.

The patient's general condition is bad despite the fact that he has so little fever. One of the very commonest things to happen and which is the final chapter in such a case is for a miliary tuberculosis to develop on top of the tuberculous peritonitis, the final picture being obscured by the symptoms of this generalization.

Note.—This patient died a couple of weeks after he was shown in the clinic. The further course of the disease was uneventful as far as any new symptoms were concerned. He grew weaker day by day and died of exhaustion. An autopsy was obtained, with the following results:

Anatomic Diagnosis.—A diffuse fibrocaseous tuberculosis of the peritoneum with obliteration of the peritoneal cavity; caseous tuberculosis of the mesenteric, periaortic, peri-iliac lymph-glands; old caseous tuberculosis of both lungs (slight) and of the tracheobronchial lymph-glands; tuberculous obliteration of the left pleura.

These findings are self-explanatory. The tuberculosis of the left pleura was old and not very extensive, which probably accounted for the fact that we were unable to demonstrate it, although we looked for it. The lung findings were old and fibrous and accounted for the difficulty in finding tubercle bacilli.

CASE III. TUBERCULOUS PERITONITIS (ASCITIC TYPE)

The third case which I wish to present is that of a Bohemian laborer, forty-nine years of age, who came into the hospital complaining of the following symptoms:

Present Complaint.—Abdominal pain, nausea and vomiting, loss of appetite, loss of weight, constipation.

Because of the patient's inability to speak much English this history has been acquired on the instalment plan and mostly from relatives. The abdominal pain seems to date back four

months, at first dull and aching, later sharp and more severe. The pain comes and goes, and on some days he is entirely free and very comfortable. The pain was located around the navel and in the right lower quadrant. He has a sense of abdominal distention and a feeling of fulness has been frequently associated with abdominal pain. Nausea and vomiting occur particularly after eating breakfast, usually twenty to thirty minutes after finishing the meal. He seldom vomits after other meals, but may feel nauseated. A loss of weight has been noted by him and he says it has been considerable, but he does not know how many pounds.

General and Negative Symptoms.—Cardiovascular symptoms entirely absent. Under respiratory symptoms we note that he has had cough and an expectoration of a thin, scanty, mucoid material. Genito-urinary symptoms are entirely absent.

With the exception of headaches we note no nervous symptoms.

His habits show only a moderate degree of alcoholism and he does not smoke.

Physical examination shows a man forty-five or fifty years of age, somewhat emaciated, and apparently not acutely ill. His general condition is good. He was afebrile on admission, pulse was 76 and respirations 20. Blood-pressure 120/80.

The *head* and *neck* present no abnormalities. Teeth are in the usual poor condition of these patients, and his tonsils show an old infection with very fetid breath.

Chest.—Expansion is good, regular, and equal. The muscles of the chest are slightly atrophic, but not more so than would accord with the general body emaciation. Breath sounds are roughened at the right apex and a few crackling râles can be heard.

Heart.—Apex-beat is in the normal position, no enlargement; no adventitious sounds.

Abdomen.—The right half of the abdomen is held somewhat rigid. There is a localized tenderness to a considerable extent noted over the right lower abdomen, to a less marked extent over the rest of the abdomen. Palpation suggests a moderate-sized

tender mass in the appendix region. Liver and spleen cannot be palpated.

Rectal examination reveals tenderness on the right side, but no mass can be made out in this area.

The genitals are negative and the extremities show nothing of especial interest. Deep and superficial reflexes are present and normal.

At the time of this patient's entrance into the hospital he was afebrile, but on subsequent days he developed some fever, which occasionally ran as high as 101.5° F. The laboratory findings are very simple. The blood showed a red count of nearly 4,000,000, hemoglobin 75 per cent., with 8000 whites, and a differential count of 76 per cent. polymorphonuclear neutrophils. The sputum was scanty and was negative for tubercle bacilli, although the lung findings persisted. A test breakfast was given, which showed an achlorhydria. The stools were negative in every direction. The Wassermann was negative.

Our first impression of the case was that of a chronic appendicitis or, if you prefer, of a recurrent appendicitis. However, there were one or two things that made us somewhat suspicious, one being that the temperature, even after rest in bed, remained always between 99.5° and 101° F. Second, he had a history which dated back a considerable time and had been productive of some emaciation. Furthermore, the *x*-ray examination showed a very definite tuberculous involvement of the upper right lobe, with very extensive fibrosis. In view of this fact we had to give serious consideration to the possibility of its being a localized tuberculosis, and in view of the further fact that the region of the appendix and cecum was always the most tender, this seemed the probable offending organ. We therefore made a tentative diagnosis of tuberculosis of the cecum or appendix, or both.

The patient seemed to improve for a while, or at least suffered less pain, and we rather lost him from sight for a week or two, until finally one day we were struck by the increased size of his abdomen, and on going over it discovered that there was a very definite fluid exudate therein. This fluid moved freely with

change of position. The patient declared that he felt better and that his pains were much improved. The temperature, however, kept up, the leukocyte count remained about the same, but the exudate continued to increase, and in the course of the next two or three weeks became quite extensive. Finally, it reached the point where it became a distinct embarrassment to respiration, at which time paracentesis abdominis was performed. Several liters of straw-colored, greenish-yellow fluid were removed, with a specific gravity of 1019 and a predominantly lymphocytic type of cell. Quite strenuous efforts were made to find tubercle bacilli in the fluid by various concentration methods, but without avail. The fluid has accumulated two or three times to such a point as to render tapping imperative. After the fourth or fifth tapping the patient seemed to improve very greatly. His temperature became lower and he became stronger and clamored to be up and about. This was, however, not permitted.

In view of these findings and this course the diagnosis could hardly be in doubt for a moment. We have to do with a typical case of tuberculous peritonitis of the so-called ascitic type, which in all probability had its origin either in a tuberculosis of the cecum or appendix, or else both of these were secondary to the pulmonary tuberculosis.

Now these 3 cases which I have demonstrated show with especial clearness the three leading types of tuberculous peritonitis: first, the caseous or ulcerative type; second, the fibrous type, which is more chronic and in which there is a large amount of fibrous tissue produced, knotting together the viscera and producing tumor-like masses, and last, the type shown by this patient, the ascitic type, in which there is a large amount of fluid present with very little inflammatory reaction. I think it may be safely stated that of this latter group the majority of the cases are secondary to a tuberculosis of some of the contiguous organs, either the periaortic glands or the mesenteric glands, or to a tuberculosis of the bowel, especially the cecum, or semi-occasionally to a tuberculosis of the genitalia, such as a tuber-

culous salpingitis or epididymitis. In a general way the diagnosis is much easier when there is a large fluid exudate, since paracentesis is readily carried out and the fluid may give valuable indications. Tubercle bacilli are extremely difficult to find, although I have repeatedly been successful in doing so. With small amounts of fluid great care must be taken in performing paracentesis, since the presence of adhesions may cause the trocar to perforate the bowel. This happened to one of my interns a few years ago, but, aside from a fecal fistula lasting a few weeks, no serious results ensued. Animal inoculation is more promising, but requires more time than is usually available.

I hope these cases will impress upon you the frequency of this disease. Some writers on the subject go so far as to consider the peritoneum the third or fourth most common place of localization of tuberculosis. In older children, especially in young girls, this ascitic type of tuberculous peritonitis is very common and its origin is only too often overlooked. It may be well to say a word or two about the prognosis. A study of the available statistics shows a recovery of perhaps 30 per cent. of the cases, using the term "recovery" to indicate arrested cases. In this hospital our experience is by no means that favorable, since the majority of our patients belong to the down-and-out class. In the last few years since the war the increasing number of colored patients with their notoriously weak resistance to tuberculosis makes our outlook even worse.

June 28th: I am showing you this patient again, and I am sure you will recognize him as the patient I presented two weeks ago, and in whom we made the diagnosis of the ascitic type of tuberculous peritonitis. We thought he was improving at the time we showed him to you, but our judgment was not sound, for he suddenly became much worse and developed very definite and classical symptoms—rapid respirations, slight cyanosis, fine moist râles all over both lungs, irregular fever, and rapidly progressing asthenia—and, as you will note, his condition is infinitely worse than when I showed him to you two weeks ago. He is almost too sick to examine, but the only things to be found are those which I have enumerated. They admit of

only one interpretation, namely, that on top of his old fibroid lung condition and his tuberculosis of the peritoneum he has developed a general miliary tuberculosis, and in the ordinary course of events the end should not be far off. It seems to me that this case is a particularly instructive one and illustrates the following points: First, of how closely a tuberculosis of the cecum or a localized tuberculous peritonitis may simulate a recurrent appendicitis; second, it illustrates the progress of the exudate in the peritoneum beginning with the localized condition, and last, it shows one of the more usual terminations, namely, in a general miliary tuberculosis.

Note.—Two weeks later. The patient died about ten days after being shown for the last time in the clinic. An autopsy was secured and a summary of the findings is as follows:

Anatomic Diagnosis.—Tuberculous ulcerations of the lower ileum, with extensive tuberculous ulceration of the cecum; diffuse tuberculous peritonitis with ascites; diffuse nodular fibrocaseous tuberculosis of the lungs; miliary tuberculosis, especially of the lungs, kidneys, and liver; healed tuberculosis of the tracheobronchial lymph-nodes.

It is interesting to note this latter finding, *i. e.*, of the healed tuberculosis of the tracheobronchial lymph-nodes. These were, in all probability, the original portal of entry, and in spite of their apparent healing the patient ultimately developed a tuberculosis of the lung to which the intestinal and peritoneal tuberculosis was secondary.

PRIMARY CARCINOMA OF THE LIVER

THE last patient whom I wish to show you today is a Swede, age sixty-two, who entered the hospital a few days ago with an examining room diagnosis of ascites due to cirrhosis of the liver. His history is as follows:

Present Complaint.—Swelling of the abdomen and lower extremities, which have existed for nearly a year, and nausea which has existed for nearly six months.

Onset and Course.—The patient states that up to a year ago he felt fine. About this time he got a pain in the lower part of his chest which he thought was a pleurisy. This pain is still present when he lies on the right side. The swelling of the abdomen also began a year ago. He says it disappeared at times, leaving a normal size abdomen. It would come on every week or so, last two or three days, and then go away, and he insists that it went away completely. The present swelling began about three weeks ago and has not increased in size yet, though he constantly expects it will. He does not think it is getting any larger. The swelling of the lower extremities began, he thinks, two months ago. His impression is that it began in the thighs and then went to the feet. The swelling is usually somewhat less after being on his feet for a time. The swelling of the scrotum became apparent two weeks ago.

Nausea had been present for six months. It comes on after eating and, as he expresses it, "like indigestion." As is usual with many patients, he feels sure its beginning was due to a meal of "short ribs" which he ate six months ago. He has considerable belching, but never has any vomiting.

Cardiorespiratory Symptoms.—The pain in the right chest has already been referred to. Shortness of breath was elicited, and he states it only comes on when walking. It has been present a number of months.

Gastro-intestinal.—His appetite has always been and is still good. Bowels are normal, he having neither constipation

nor diarrhea. If it were not for the swelling of the abdomen and legs he would consider himself quite well.

Genito-urinary.—With the exception of a nocturia, requiring him to get up two or three times at night, and which has been present for the last two or three months, no abnormalities could be elicited.

Family History.—Shows nothing unusual. His wife and several children are living and well, as are also several sisters and brothers. His father and mother are dead of unknown causes.

Habits.—He does not smoke, but has been a heavy beer and whisky drinker up to about ten years ago. He said he could stand any amount of alcohol without getting drunk, and feels sure for this reason that it never hurt him. However, for reasons which we cannot learn, he had decided to stop drinking, and has taken no liquor for the past ten years. His occupation is a teamster. This fits in very well with our observations in this hospital, which show that the plumbers, teamsters, and truck drivers are among the heaviest drinkers we have.

His pathologic history shows that he had typhoid and pneumonia in 1894. He denies venereal infections, and I may say that his blood Wassermann is negative.

Physical Examination.—This is identical with what it was on his entry, so I will read you the findings then:

Patient is a white male, looks to be about sixty, and does not seem to be acutely ill. Temperature and respirations were normal on admission and pulse 90. Blood-pressure was 130/80. His general appearance is slightly apathetic. He answered questions promptly and insists that he feels all right except for his swollen abdomen and legs.

Head.—Eyes, ears, nose, and throat are negative. Tongue is somewhat coated and, as usual, there are many badly decayed teeth. In general, the facies is that of a heavy drinker, which probably explains the dilated venioles on the skin of the face.

Chest.—Lungs are normal except that there is a moderate dulness at both bases. Inasmuch as the sounds over these regions are approximately normal, I think we may conclude that

this is due to the pushing up of the diaphragm as a result of the increase in the abdominal contents. The heart is without abnormalities.

Abdomen.—This is markedly distended and the skin is particularly glossy. As you see, the flanks show a distinct bulging, the so-called "frog" belly, and there is a very definite flatness in either flank, which shifts from side to side when the patient is turned on his side. A fluid wave can be readily elicited, and it is a very simple matter to demonstrate the existence of a considerable fluid exudate.

In addition to this, and, I may say, in spite of it, it is easy to demonstrate a considerable sized mass in the right upper quadrant. This mass moves freely with respiration, has a rounded edge, extends at least four fingers below the edge of the ribs, and has a very uneven surface. If we examine this surface more carefully we see that there are definite masses upon it, at least three or four in number, the smaller being apparently about the shape and size of the small end of an egg and the larger feeling like half of a medium-sized apple. Percussion shows that the liver extends an interspace higher than normal, and since, as I have already stated, it extends down at least four fingerbreadths below the edge of the rib, it is evidently very greatly enlarged. The spleen cannot be palpated, but percussion seems to indicate a moderate enlargement of the organ. The kidneys are not palpable. There is no especial tenderness over the abdomen or on pressure over the liver. The masses in the liver are not umbilicated.

Genitalia.—Normal, with the exception of a considerable edema of the scrotum.

Reflexes show nothing abnormal.

Extremities.—Both extremities are markedly enlarged and pit readily. The edema is a soft one, which speaks for its recent origin.

Perhaps it will be instructive before we take up the laboratory findings to consider the possibilities in this case, and see if we can come to any conclusion. To begin with, we have a history dating back at least a year. The patient is intelligent

and I am inclined to accept his statement, at least so far as he is competent to judge, when he says that the abdominal swelling came and went, or at least grew larger and smaller in the beginning. My experience is that an individual of his mental caliber would notice an edema of the legs much more quickly than he would a moderate swelling of the abdomen, so when he assures us that the swelling of the abdomen preceded that of the legs by six months, I am inclined to take his statement at par. Furthermore, our physical examination has shown no cause for a circulatory edema of the legs except that of an intra-abdominal pressure, which is more than adequate to cause it. I think we may then safely say that the intra-abdominal pathology is the primary cause and probably the whole of the trouble. With a man of his habits, having been a hard drinker, the first thing that would cross our mind would be an alcoholic cirrhosis of the liver, and, if you remember, that is the diagnosis which was made in the examining room, and is also the junior intern's diagnosis as recorded on the history. Let us consider this a moment. The man states positively that he has not touched a drop for ten years. Would that invalidate a diagnosis of alcoholic cirrhosis? I think not. It is a fairly common experience or, rather, it has been a fairly common experience, for cirrhosis of the liver is no longer very common, to find that a patient for some reason or other stops drinking, and yet several years after may succumb to an alcoholic cirrhosis. I judge that the explanation is that the alcohol or possibly an associated infection sets in motion chronic degenerative and inflammatory processes which are too wide-spread to come to a standstill even though the drinking was stopped. Now it is not always thoroughly appreciated that most cirrhotic livers, at least as we see them here, are large. The classical gin-drinker's liver has been so often pictured as very small that many clinicians fail to comprehend that this is not the normal state of affairs for a cirrhosis. As we see them clinically, more patients with cirrhosis die with the liver larger than normal than with a small liver, and my pathologic colleagues have confirmed this statement. One of them goes so far as to say that three-fourths of the cirrhotic

livers which come to necropsy are larger than normal. The size of this liver, then, is no obstacle, but when we come to study the surface we meet with a very distinct obstacle. As you know, the granulations of even a markedly hob-nailed liver are too small to feel through the abdominal wall. Generally, when one feels granulations they are really in the abdominal wall and not in the liver. What, then, are these definite masses? Our minds at once revert to another type of cirrhosis, namely, the syphilitic, the so-called botryoid liver. In this type of liver we have not gummata themselves, but the result of gummata which have healed out and left thick strands of scar tissue which cut up the liver into many pseudolobes, and these, when felt through the abdominal wall, can readily enough impress one as being hard tumor masses. The mere fact that a man has been a hard drinker does not preclude his cirrhosis being of a syphilitic type. However, we find a negative history of syphilis. We have not been able to find any scars of a primary lesion and the Wassermann is frankly negative, so that this diagnosis is hardly tenable. We shall have the Wassermann repeated to make sure. A liver of this kind is, however, much more frequently due to metastatic carcinoma and, just offhand, this is the thing which should engross our attention. To be sure, we have no symptoms which really point toward any other organ as a probable seat of a primary tumor. The nausea he complains of might possibly point toward the stomach, and there is this to be said in favor of primary carcinoma of the stomach, namely, that it is perhaps of all the intra-abdominal carcinomata, the most frequently "silent," that is to say, a tumor of the stomach away from the pylorus may exist with almost no symptoms whatever, and may go on to produce, literally, pounds of metastases in the liver before the stomach condition is even suspicioned. We have gone into the stomach condition quite carefully and find the following: The Ewald test-breakfast removed one hour after gives 75 c.c. residue, with a free acidity of 38 and a total of 59—slightly higher figures than normal if anything. This was a little of a surprise, because, as a rule, men who have been hard drinkers have some degree of chronic gastritis with a lowering of the acidity. No

blood or lactic acid have been found. In regard to the examination of the stools, they have been absolutely normal in every way. The color has been yellow and bile has been demonstrated in normal quantities. The *x*-ray examination of the stomach was frankly negative. This has been done twice, and nothing can be determined of an abnormal character. At the second examination particular attention was paid to the duodenum, having the idea in mind that we might be dealing with a primary carcinoma of the pancreas which sometimes presents an abnormality in the duodenum. Nothing could be found here. From the clinical side, a primary carcinoma of the pancreas, especially if it does not involve the head, is, in my experience, one of the most difficult to diagnose. It has, however, one symptom which is almost constant, that is, deep-seated, almost continuous pain. I have learned to place more stress on this symptom, especially in the beginning, and the complete absence of pain in this case leads me to believe that carcinoma of the pancreas is exceedingly improbable. I think we may go a little further and say that inasmuch as this abdominal condition dates back at least a year, it would be almost inconceivable for a carcinoma of the pancreas to have existed so long and to have made such extensive metastases without producing a jaundice as the result of the obstruction of the common duct. I am strongly of the opinion, therefore, that we have not to do with a primary carcinoma of the pancreas and its metastases.

We next think of the other portions of the gastro-intestinal tract, and these may be rapidly disposed of. The *x*-ray, both the barium meal and the barium clysma are completely negative, and repeated examination of the rectum shows nothing abnormal. I have already mentioned that no blood was to be found in the stools, so that we have no reason at all for considering a primary carcinoma anywhere in the gastro-intestinal tract. The same reason applies to the prostate. While this organ is perhaps a little firmer than normal, it cannot be called hard, and is certainly not nodular, so we shall have to dismiss it as a seat of the primary tumor.

The same reason applies to the bladder. Cystoscopic exam-

ination shows nothing out of the way, and the urinalysis is quite negative, merely a trace of albumin which one would find in almost any patient of this age.

This narrows the field down very considerably. We might think of a malignant neoplasm of the kidney, although the majority of these are hypernephromata and produce their metastases in the lungs rather than in the liver. No masses can be felt in the flanks and, as I have stated, the urine is practically negative. The phthalein output is approximately normal and the blood chemistry is normal for a man of his years. Indeed, his functional kidney tests are rather better than usual. The negative findings in all of these directions leaves us rather hard put to it for a diagnosis. To be sure, carcinomata occasionally occur in out-of-the-way places, such as bones, etc., but we have not a single symptom or sign to justify any such assumption. The thought occurred to me that it might just be possible that it was some unusual granuloma. The blood examination reveals, however, 4,800,000 reds, 10,400 whites, with a substantially normal differential picture. So here again we have no data on which to base such a diagnosis.

I think, now, we must ask ourselves the question as to whether, after all, we really have do do with a carcinoma in any form, and we have to consider some of the other rare conditions in the liver. An echinococcus cyst occurs to me particularly, because I have under observation now a woman in whom I have made this diagnosis. However, this patient has lived a great portion of her life right here in Chicago, has never visited any parts of the world where an echinococcus would be likely to be acquired, and then, too, the tumors have presumably grown within the last year, and that is hardly to be expected in a cyst of this sort. We have no eosinophilia, which is another point which may be considered. However, to make matters as sure as we can, I shall have the complement-fixation test for echinococcus done. The last case I saw of this disease in which the tumor was in the lung gave a very beautiful positive test in this matter, which was afterward confirmed by the patient's coughing up some of the cyst membranes. If we sum up the

situation as it stands now, unless the complement-fixation test for echinococcus proves positive, we are face to face with this group of facts: We have multiple hard masses in the liver which have grown in the last year, which are apparently malignant, even though not as malignant as sometimes found, and for which we can find absolutely no primary source, and this opens up the question as to whether we may be dealing with one of the extremely rare cases of primary carcinoma of the liver. This is a diagnosis which one hesitates to make for the very adequate reason that when made it is wrong about three times out of four. In spite of the utmost care there is apt to be a primary tumor somewhere else. I must confess that up to the present time we have singularly failed to locate or even suspicion any other source than the liver. The rarity of primary carcinoma of the liver is such as to make one think a long time before risking such a diagnosis, for the odds are enormously against one in so doing. I think, perhaps, we will not discuss the case any farther today, but will wait until next week and watch developments. I shall go over him again carefully and, in particular, have all the laboratory work repeated, and see if anything comes of it.

Clinic One Week Later:

You will recognize, I am sure, this patient as the one whom I presented to you in this clinic a week ago. So far as the examination is concerned there is not a great deal to be added. The patient still complains of little or no pain and states that his only trouble is that his abdomen feels full. There is not the slightest tinge of jaundice and the urine has been repeatedly examined, with negative results, for bile. You will remember I told you how difficult the diagnosis of carcinoma of the pancreas might be, but I felt we could reasonably exclude it because of the complete absence of pain. Nevertheless we have had some of the tests for pancreatic ferments in both duodenal contents and stools carried out, with, however, entirely negative results as far as indicating carcinoma is concerned. I have had another Wassermann made, which again was negative. The stomach

and duodenum has again been subjected to fluoroscopic examination, also with negative results, and the stools continued to be negative for blood or other abnormal constituents. There is perhaps one other organ which might be considered as a possible primary source of carcinoma, and that is the gall-bladder itself. I have seen cases in which the extreme tip of the gall-bladder only was involved with a liver full of metastases. Now in this particular case we have no history of a previous gall-bladder infection or of a cholelithiasis, and while it is almost impossible to exclude these with certainty, yet we have no reason to make such a diagnosis. You can see, therefore, in spite of everything we can do to find the primary focus, we have utterly failed to do so. Perhaps you may wonder at my reluctance to make the diagnosis of a primary malignant disease of the liver, but when you consider that probably not more than 2 or 3 per cent., that is, in from 1 to 30 or 1 to 50 of the cases is a carcinoma of the liver primary, you will better comprehend my reluctance. Having failed, then, to establish any other focus than the liver as a starting-point, and being convinced in our mind that the tumor is malignant, let us look at it from that standpoint for a moment. Might it be a primary sarcoma of the liver? Yes, it might, but this tumor is even much more rare than a primary carcinoma, grows much more rapidly, causes much more pain, and occurs in younger individuals. I have never seen but 2 cases, one in Weichselbaum's Pathological Institute in Vienna, many years ago, and the other in this hospital some five or six years ago. Have we any evidence which might be adduced toward making the diagnosis of primary carcinoma more probable? I think we have. To begin with, the patient was a heavy alcoholic consumer for many years, and this, as you well know, predisposes strongly toward cirrhosis. Now it is a fact that most of the carcinomata of the liver are associated with cirrhosis, and some of them in a causal way. I refer in this latter group to the so-called cirrhosis carcinomatosa. In general, we distinguish three groups of primary cancer of the liver—first, the nodular type; second, the massive type, which consists of one enormous mass, and lastly, the cirrhosis

carcinomatosa, in which the carcinoma starts in innumerable spots more or less simultaneously. This is the type of liver to which the Germans give the name "Leberzellenkrebs" or "liver-cell carcinoma." In this particular type, which should be regarded as the true carcinoma of the liver as distinguished from carcinoma of the larger bile-passages, the disease begins with a picture of cirrhosis, and the liver may undergo even a shrinkage in size, and then suddenly begins to enlarge and generally to become painful. It seems to me that the unusually long duration of this case, for, as you will remember, we have definite signs of enlargement of the abdomen for a year, may be utilized with some reservations as pointing in this direction. So on the basis of the previous history of alcoholism, the slow and steady growth of the liver tumors, the absence of jaundice, and the absence of every other symptom or sign to incriminate other organs, I am going to venture the diagnosis of primary carcinoma of the liver, probably of the cirrhosis carcinomatosa type.

I have suggested an exploratory incision to the patient with the idea that it might possibly be some other condition capable of being operated, but he declined.

Note.—This patient died five weeks after the above clinic was taken down. There had been no appreciable change in his condition except that a considerably greater degree of ascites had developed and a slight icteric tinge to the conjunctiva. My intern has a note expressing his surprise at the absence of all pain and serious discomfort, and his general feeling of cheerfulness up to a few days before death. I had just left for my summer vacation a day or two before the patient's death, but through my intern's courtesy I have a synopsis of the pathologic findings at autopsy.

Anatomic Diagnosis.—Primary carcinoma of the liver. The liver was almost completely replaced by carcinomatous tissue of apparently the same age everywhere. Indeed, it was impossible to determine whether or not there were any cirrhotic changes present, but some could be seen in the left lobe, which was not so greatly affected. A most careful search failed to reveal any primary focus. At the present writing the histologic study of

the tumor is not complete, but from the clinical standpoint this interests us to a much less degree than does the question of the primary nature of the tumor. I wish to repeat again the great rarity of these cases, and I feel quite sure that some of the reported cases are perhaps not genuine, and that a small carcinoma perhaps of the prostate or some out-of-the-way organ has

Fig. 107.—Primary carcinoma of liver. Note the manner of distribution of the tumor masses, beginning in a large number of places simultaneously, and each nodule shading off gradually into the normal liver tissue. This is in sharp contrast to the sharply defined nodules in metastatic carcinoma. Although the liver was greatly enlarged, there was some evidence of cirrhosis in the left lobe. The general shape of the liver is fairly well preserved.

been overlooked. This is only the second case that I have seen in several years, and the last case I was not sufficiently sure to hazard the diagnosis.

One of the gentlemen reminds me that we overlooked one point which might have shed some light on the subject, and that is that we might have done an abdominal paracentesis, and the nature of the fluid might have shed some light upon the disease.

I plead partially guilty to the indictment, but the amount of fluid was small, and I hesitated to do so under those conditions. In my experience where the peritoneum itself is not involved the fluid obtained is an ordinary transudate, and will have substantially the same characters, both physical and chemical, whether the underlying cause be obstruction from cirrhosis,

Fig. 108.—Cut section of liver shown in Fig. 107. The enormous number of nodules of approximately the same size is well shown.

from carcinoma, or from the pressure of glands in the portal fissure. As a matter of fact, my contention is sound, since some fluid was removed at autopsy and examined and showed the usual characters of a transudate, with low specific gravity and a relatively small number of lymphocytes present. In other words, the ascites was a mechanical affair and in no way dependent on the nature of the obstruction.

CLINIC OF DR. JOSEPH C. FRIEDMAN

MICHAEL REESE HOSPITAL

THE INTERPRETATION OF PROLONGED MINIMAL TEMPERATURES

THE first patient representing this group which offers considerable diagnostic difficulty has been followed by me at intervals since 1913. At that time she complained of indefinite gastric disturbances about one hour after eating, relieved temporarily by food, without pain and without nausea, with constipation and temporal headaches at intervals. She had always been healthy except for repeated nasopharyngeal infections, for which tonsillectomy had been done eight years previously.

At the first examination we found a well-built, stout young woman, thirty years of age, with completely normal findings except for a slightly impaired resonance at the apex of the right lung posteriorly, but with no changes in the breath sounds and no râles. On an anticonstipation diet she apparently improved and was not seen again until October, 1916, when she returned, complaining of pain in the right hip and at times swelling of the right ankle. The pain was worse on sitting down and when tired, but not on walking. At times it was felt in the back, and her constipation had again become pronounced.

There was a slight afternoon temperature, 99.6° to 99.8° F. Her pulse in the office ranged from 86 to 90. There was some tenderness in the right iliac fossa, apparently of the sacro-iliac joint, and in the skin of the upper, outer side of the right thigh.

Her reflexes were normal. There was no atrophy; no swelling of the ankles was visible. There was bilateral, moderate flat-foot. White count was 8800. Urine was normal. *x*-Ray of the femur and hip was negative. The diagnosis at this time was some chronic infection located either primarily in the pelvis

or metastatic there, and involving the lumbar plexus in the external cutaneous branch.

The above attack was typical of a number of others which she has had at intervals since then. For instance, in 1919 she complained of a cough, felt tired, and on examination there was impaired resonance at the left apex posteriorly to the midscapula region. At that time numerous small râles were heard over this area. She had pain in the right shoulder, right leg, and lower abdomen as well, and a constant feeling of exhaustion. There was no frequency of pain on urination. There was tenderness over the right thigh, and at this time it was so pronounced that a surgeon called in consultation believed that there must be a subperiosteal abscess and advised operative interference. This was done, but no inflammatory focus found.

As usual, during the exacerbation of her infection she was constipated, had tenderness along the colon, and the stools showed marked increase of mucus. There were a few pus corpuscles in the urine, but cultures were negative. White count was 12,300. The findings otherwise were normal except for the same slight, continuous temperature which in the hospital attained a maximum of 99.2° F.

In view of the negative findings in the femur, the two possible sources of infection at this time were the lungs and the colon. The pulmonary infection was definitely non-tubercular, while, of course, the possibility of infection resulting from the slight colitis could not be ruled out. After a prolonged rest in the country there was considerable improvement. She was able to return to her work.

In 1921 there was again a relapse, the patient stating at this time that there was slight loss of weight, that she had frequent colds, which she described as "slight attacks of pharyngitis," followed by a recurrence of her subfebrile temperature, which was rendered apparent to her by a slight afternoon flush. In this manner a rise of temperature to 99° F. was distinctly felt by her and could always be verified by the thermometer. This particular patient felt the flush in her cheeks. In others it was subjectively more marked in the ears.

In this attack, though the abdominal pain was not pronounced, there was very marked tenderness over the right sacroiliac joint. On *x*-ray examination of the appendix it was mobile, though the cecum was quite tender. Again, on advice of counsel, an operation was done in hope that the source of the colon infection might reside in the appendix. A laparotomy, however, revealed a perfectly normal organ, which was removed. Again, after a prolonged rest, the temperature, which had ranged from 99.6° to 100° F. daily, subsided and remained normal except after considerable exertion.

She felt well for a considerable period except when she would acquire an acute pharyngitis, when a prolonged slight rise in temperature, with the same type of pains, would recur. During these years vaccines had been made from the sputum on two occasions, and two prolonged courses of therapeutic injections given, without any result. It is needless to say that the usual causes of slight temperatures were considered and ruled out. Her basal metabolism was normal and, as stated above, no evidence of pulmonary or other tuberculosis, and no marked degree of dental infection, could be determined.

Following her last series of infections she was sent to the Southwest and remained there two years, returning in June, 1923. During the last year of her residence there she had no upper respiratory infections, and since then her temperature has been normal, with an absence of the accompanying phenomena of pain and exhaustibility.

In an attempt to diagnose this case all the more easily accessible sources of infection were carefully examined. As has been stated, tonsillectomy had been done several years before. The sinuses were repeatedly and carefully examined, without result. The teeth were carefully gone over, more as a tribute to the present dominant theory of their importance in such cases than as a result of our own experience, which has been that they play an extremely secondary rôle in the general bodily condition, and, when found diseased, are far more often a result of general ill health than a cause for it. This, of course, is only an opinion, but, I believe, is becoming gradually more prevalent.

Another greatly overestimated source of chronic infection is the appendix. In our own experience chronic appendicitis, in the strict sense, meaning a condition of chronic inflammatory change without acute exacerbation, is a very rare condition. To be sure, repeated acute attacks are frequent, but this, of course, is a different entity than that above mentioned. The removal of the appendix in this case, in an attempt to discover the source of the infection, was about as successful as this procedure has usually been in our hands.

Mild grades of chronic colitis are also very infrequent causes of temperature. Of course, chronic ulcerative colitis with accompanying destruction of the mucosa is a frequent source of bacterial infection and intoxication, but such is the resistance of the intestinal mucosa that actual destruction of the mucosa must take place before living bacteria can gain entrance into the lymphatics over a prolonged period.

Far more frequent are mild infections of the kidneys with accompanying bacilluria, even without pyuria. The bacilluria, of course, is of the colon type as a rule, and, therefore, easily demonstrable on culture. It was absent here.

There was left, then, the infections of the upper respiratory tract. In substantiation of this theory was the presence at different times of cough and finding of râles in the lung. At one time a friction-rub was present in the left axilla, and the fact noted by the patient herself that her febrile periods were generally preceded by a slight pharyngitis. It was, to be sure, of an extremely mild type, ephemeral, occasionally accompanied by enlarged and tender cervical glands.

When sent away into a sparsely settled district, where no throat infections occurred, she apparently recovered, and on this evidence the final diagnosis remained of recurrent pharyngeal infection with cervical and peribronchial adenitis. Pains which had misled us so frequently were simply toxic phenomena of the primary focus.

There are, I believe, two lessons to be learned from this case which is representative of a considerable group in ambulatory practice. The first is that the source of a chronic infection may

be in an organ in the deeper lying structures of the body. For instance, in a number of attacks in this patient the cervical glands were slightly enlarged. In other attacks the cervical glands were not enlarged. There was no evidence of the lung parenchyma being involved, and yet the febrile disturbances and the resulting toxemia was very evident. What more logical than to assume that the deeper glands of the chest had become involved and were now the seat of bacterial activity? We are still, unfortunately, so much the victims of habit that we are prone to believe that when tonsils, teeth, and perhaps gall-bladder, appendix, and genito-urinary tract have been ruled out, that no other seat of chronic infection remains. Such is not the case, even though these other sources of infection are extremely difficult to demonstrate. I have already mentioned my own firm conviction of the minor rôle played by teeth in this connection.

My attention has frequently been attracted to a second phenomenon noted here, and that is the apparent tendency for throat infections in tonsillectomized patients to cover a wider area, and even when mild to spread rapidly into the deep cervical glands. In other words, the tonsils are really barriers to the spread of infection, and when removed render the deeper structures more vulnerable. In the above case the temperature rise was up to 100° F. at frequent intervals and, therefore, there could be no question that the rise was a pathologic one. There are, however, a considerable group of patients in whom the maximal temperature may be 98.8° to 99.8° F., even on repeated examinations. For instance, in 100 cases, taken at random from an office file, 48 were within those limits. In such cases when are we justified in considering such rises within normal limits and when pathologic? The question of the maximal normal temperature is somewhat in doubt, as the means of determining it have varied. For instance, Benedict used rectal temperatures only, Wunderlich, axillary temperatures. However, the preponderance of authority seems to be in favor of considering 99.1° F. as the maximal daily norm in individuals moderately exercising. In the above 48 cases there were 18 between 98.6°

and 99.1° or 99.2° F., and 30 above the latter point. On the whole, the 18 below the dividing line exhibited far less pathology than those above, consisting mainly of neuroses of various types, though, of course, there were occasional patients with more severe ailments, such as encephalomalacia, etc. As far as such a limited series is worth anything as evidence, 99.1° or 99.2° F. is a reasonably accurate dividing line between the normal and clearly abnormal cases. Variations from the normal, however, need not necessarily be due to bacterial cause, and apparently the type of toxin producing them may be one of a large number because of the rôle played by the nervous system in the regulation of body temperature.

This rôle is evidently an important one, as shown in the experimental work on poikilothermic animals, *i. e.*, animals in which the heat regulatory mechanism has been temporarily suspended. In them infections produced no increase of metabolism, showing that the increase in metabolism in febrile infections is mediated by the nervous mechanism for temperature regulation, and not by direct action of the infecting agencies on the tissues themselves. Any agent, then, which affects this nervous mechanism or apparently parts of the central nervous system is capable of causing variations in temperature.

Again, since the temperature norm is a result of heat loss, as well as heat production, and heat loss is apparently directly related to the state of the skin capillaries, agents which affect the vasomotor system are able to cause at least slight rises in temperature. The result of all this nervous mediation is the very frequent occurrence of slight rises in temperature in the patients with apparently no other condition but a functional nervous disorder. This, of course, excludes mild cases of Basedow's, and includes that condition known as irritable heart in which such rises of temperature are so frequently observed.

The following history illustrates a case of this sort: A patient, twenty years of age, complained of pain in the precordium, going through to the back and along the left hypochondria to the lower axilla. It was brought on by exertion and from running upstairs or by any moderate exertion, such as sweeping, after which

she felt some palpitation, slight dyspnea, and some exhaustion. She claims to faint during the attacks, but has never lost consciousness. She frequently has subjective dizziness, mild headaches, especially on bending over. Her appetite has always been poor. She sleeps fairly well and is extremely nervous. She had a moderately severe attack of influenza about a year ago, but her present symptoms preceded this. Her mother died of heart trouble at thirty-three. One sister has "enlargement of the heart." There are 3 brothers living and well. On examination she was fairly well nourished. Blood-pressure was 110 systolic, 75 diastolic. Pulse was 108. Temperature was 99.4° F. by mouth. Heart was normal. Lungs were normal. Thyroid was just palpable. There was no tremor, exophthalmos, or Graefe's. Abdomen was normal. Reflexes were normal.

The following afternoon her temperature was 99.2° F. and pulse 84. In the first day her pulse of 108 could be brought down to 76 by taking a deep inspiration. On exercise the heart regained its initial rate within two minutes. She was sent to the hospital for observation. *x*-Rays of the chest revealed no abnormalities. No râles were heard. Basal metabolism was normal. Blood examinations were normal. Her temperature from October 19th to 26th, with the patient up and about the ward, ranged from 99° to 99.6° F. daily. From October 26th to November 4th, the date of discharge, it was never above 98.6° F., and has remained this way to the present time.

There were two sources of psychical trauma present. The first was the prolonged illness and death of the mother from heart trouble and the prolonged illness of one sister from some type of heart trouble. The second was her enforced residence, after the death of her mother, with an unsympathetic married sister who required her to do considerable housework on her nightly return from a hard day's work in an office. After assurance that her heart was perfectly normal and advice to change her home, with a brief stay in the hospital to recover her nervous equilibrium, the temperature ceased permanently. No other therapeutic measures were employed. None of the usual sources of infection could be determined, and yet this patient ran a tem-

perature, under careful observation, which was not due simply to the excitement of an office examination, but persisted for eight days in the hospital. Nor can I believe in any other explanation for this subfebrile rise of temperature than disturbance of the heat-regulating mechanism due to psychical trauma. The diagnosis was neurasthenia, and is, I believe, justified by the subsequent course.

We know, of course, that exertion, if sufficienty prolonged and violent, is capable of causing rises of temperature of 1 or 2 degrees. It is equally true that in pathologic conditions, such as tuberculosis, very slight exertion may cause slight rises of temperature, but it is also true that even in normal individuals, probably with unstable vasomotor systems, even the slight exertion involved in visiting a physician's office or possibly the attendant excitement may cause such a rise.

The following abstract of a history is an example: The patient is a young married woman of twenty-seven. She had been under observation the previous year for subacute appendicitis, during which time she had been taught to take her own temperature. This year she came down with some abdominal pain which pointed toward a mild inflammatory condition in the transverse and sigmoid colon. She recovered promptly from this and visited the office on several occasions, and at each visit her temperature was found to be between 99.2° and 99.4° F., in spite of her insistence that she felt perfectly well. She was requested to take her temperature at home and record it. It was then found that her home temperature never reached beyond 98.6° F., although employed as a maid doing considerable work, while in the office it was invariably high. She was quite an intelligent observer, and said that she felt on coming into the office that her skin was warmer than at home. There is no reason, I believe, to look further for this abnormal rise than simple vasomotor instability.

Another cause for slight rises in temperature is still undecided, and that is menstruation. It is quite certain that in the normal woman menstruation causes no rise in temperature, but it is, I believe, reasonably certain that in certain mild or pronounced

pathologic conditions a premenstrual rise—*i. e.*, a temperature about 99.1° F.—occurs. Again, reverting to abnormalities found in tuberculous patients, Turban determined that in early tuberculosis in 73 per cent. of the cases from a few days to a week before menstruation there was a rise in temperature from 99.5° to 100° F. in patients who had had none at all previously, while in those patients who had been having slight temperature there was a premenstrual exacerbation. Kraus and others have verified this. Quite similar to the rise in exertion and excitement of patients with an irritable nervous system there is apparently a premenstrual rise in such patients. The following is an example of such a case, with some interesting complications:

The patient was a married woman, thirty-six years of age. She had always been quite healthy, but in the last two years had indefinite abdominal complaints and was probably exhausted from unusually severe strain at home. Her condition was considered to be purely functional. The pains complained of were in the hypochondria, both iliac fossæ, simply the result of colon spasm incident to a chronic constipation. She was seen over a period of six years, during which time it was noted that on the three or four days preceding menstruation and in the first three days of the period temperatures up to 99.2° F. were observed, while after that time the maximum was 98.6° F. At one time during our absence from the city she complained of pain in the right iliac fossa. A temperature of 99.2° F. was found, the *x*-ray examination repeated, some supposed pathology in the appendix discovered, and the appendix removed on the basis of pain, temperature, and a very slight elevation in white count. As nearly as could be determined from her description of the operation, the appendix was normal. At any rate, the same attacks continued after the removal of the appendix, and it is very evident that her temperature, which was the deciding factor in advising operation, was simply her usual premenstrual rise.

She, of course, was a psychasthenic, and this, I believe, was a pathologic factor in determining this premenstrual rise, as is tuberculous intoxication in other cases. I believe I have

observed this same premenstrual rise in other conditions which would ordinarily proceed with normal temperature.

For instance, in the young woman twenty-two years of age, with an old endocarditis of the mitral valves and moderate decompensation, it was observed that with complete rest at home at the beginning of her illness her temperature from November 23 to December 14, 1922 never exceeded 98.6° F., that at this time it rose from 98.8° up to 99.2° F. and continued until December 17th. When she stopped menstruating her temperature again became normal, while in the following month she had a temperature of 99.2° F. for three days preceding menstruation. This, of course, may be a coincidence, but, at any rate, was the only rise observed during her illness.

Other long-continued, slight rises in temperature cannot be definitely classified. For instance, a young married woman of twenty-five, seen first in September, 1923, had been complaining for some months of cough non-productive in character, slight sore throat, and feeling of exhaustion. She had had influenza three years previously, after which she felt below par for two years. She then had some abscessed teeth removed, and claimed to feel better. At that time she weighed 109½ pounds, had pulse of 108, temperature of 100° F. There was some impaired resonance in the right apex posteriorly, but no râles, a slight, soft systolic murmur in the second left interspace, with normal heart findings. There was no expectoration. There were no signs of hyperthyroidism. *x*-Ray of the lungs revealed no active pathologic lesion. Basal metabolism was 1.4 per cent. above normal. Blood Wassermann was negative. White count was 8600, hemoglobin 80 per cent.

On the advice of a specialist the tonsils were removed. At a recent examination her weight was 108½ pounds, temperature 99.6° F. She was still tired and nervous. The heart findings were as before, and it is possible that during this period of over a year she was having a very mild endocarditis without embolic phenomena. The picture of that condition is, of course, very different from the usual acute bacterial endocarditis, or even from the rheumatic endocarditis with its accompanying ar-

thritis, and, as in the present case, resembles more a mild tuberculosis or an exophthalmic goiter.

Extremely difficult to diagnose are those complications of functional and organic disturbances with accompanying minimal rises in temperature, as in the case of a patient, a woman aged thirty-eight, seen in November, 1921, who complained of pain in the epigastrium, which was rather a severe pressure than an actual pain, extending up under the sternum, some dyspnea on exertion, a burning sensation in the back and in both hypochondriæ, worse one to two hours after eating, but without nausea or vomiting, and occasionally attacks of dizziness. She had had influenza twice, the first three years ago, and following that her first attack of stomach trouble. She had 2 children, 2 miscarriages. One child was subnormal mentally. Her husband gave a history of luetic infection some years before marriage. Her reflexes were normal. Heart outline was normal, and there was rather a short, harsh, systolic murmur in the aorta, loudest in the left fourth interspace near the sternum, not audible at the apex, aortic second sound slightly accentuated. She had marked tenderness in the midepigastrium, none in the right hypochondrium, also tenderness over the tenth dorsal vertebra. Physical examination was negative except for the temperature, which ranged from 99° to 99.4° F. Because of her subnormal child she had been under a severe mental strain for a number of years. Blood Wassermann was negative. Her gastro-intestinal *x*-ray showed slight duodenal stasis and a typical gall-bladder shadow which we considered insufficiently definite to be the evidence of an overfilled or a thickened gall-baldder, *i. e.*, of a pathologic condition. Her temperature continued.

One might be satisfied in calling the condition a cholecystitis and attributing the rise in temperature to that, but in our experience simple, chronic cholecystitis of the mild grade which this would be is rarely accompanied by even slight rises in temperature, such rises being limited to those cases in which the inflammatory process is more severe and more acute. It was believed here that she had more probably a luetic aortitis with involvement of the aortic valve, and that this was the cause of the

febrile disturbance. She was given antiluetic treatment, and in November, 1923 her temperature became normal, and has remained so since. The gastro-intestinal disturbance continues to the present time to a slight degree, not enough, I believe, to warrant operative interference in the presence of the cardiovascular disturbance.

To sum up, then, temperatures above 99.1° F. by mouth are abnormal. They may be indicative of well-defined pathologic processes, such as infections or other toxemias, or, on the other hand, they may be the result of an abnormal nervous mechanism acting in conjunction with a physiologic process, such as menstruation.

A CASE OF PNEUMOLITHIASIS

THE patient is a man about sixty years of age who has been observed by me at intervals for nine years. The history divides itself into two parts, the first from 1915 to 1921, and the second, which interests us most, from 1921 to date.

Originally he consulted me because of a choking sensation and at times precordial pain on slight exertion. The previous history was of no importance, all venereal infection being denied. He was a well-built, rather heavy man, the positive findings consisting of a moderate enlargement of the transverse diameter of the heart and hypertrophy of the left ventricle, with a very loud, harsh systolic murmur in the apex and aortic areas. The electrocardiograph showed a moderate number of ventricular extrasystoles, inversion of the T-wave in Leads I and II, slightly prolonged P. R. interval.

There was normal blood-pressure. The pupils reacted normally. The right knee-jerk was absent, the left greatly diminished. At this time the diagnosis of myocarditis, sclerosis of the aorta and mitral valves, angina, and tabes dorsalis was made. Antiluetic treatment was instituted, in spite of which the following year he began to complain of pains, sharp and shooting in character, in the right thigh and knee, coming on at intervals. Under antiluetic treatment and careful regulation of his mode of living he improved considerably, and the years 1920 and part of 1921 were quite comfortable.

He was seen again in June, 1922, when he gave the following history: In December, 1921 he had sore throat, pains in the joints, temperature of 101° F., and was sick ten days. He had some cough, moderate amount of expectoration, but not bloody. The attack, though mild, left him with a choking sensation and a chronic cough, symptoms which had not been present before. He had a second attack in April, 1922, at which time he had a chill, followed by a temperature of 104° F. He was then sick about four weeks, with considerable cough and wheezing. During the

next six weeks he had three more attacks of a few days' duration each. He had constant dyspnea in the intervals with orthopnea at night.

On examination (June 21, 1922) his pupils were small and reacted normally. The knee-jerks were as on the previous examination. There was no tenderness of the precordium, no tracheal tug. A slight pulsation was visible in the second left interspace. The radials were equal. Pulse was 87, with occasional extrasystoles. Heart oulines were the same as on the previous examination. The systolic harsh murmurs were present as before. There was a markedly diminished excursion in the left upper chest. The lungs were resonant on percussion. There were diminished breath sounds and absence of tactile fremitus over the entire left side, normal findings on the right side. Blood-pressure was normal. The *x*-ray report at this time is as follows: "The films of the chest show the left lung quite emphysematous in comparison with the right. Under the fluoroscope there is a slight delay in the emptying of air of the left lung in comparison with the right. The aorta is slightly broader than normal, but shows no limited area of dilatation."

It was evident at this time that there was an obstruction of the left bronchus. The first probability before the *x*-ray examination, and in view of the previous luetic findings, was an aneurysm in spite of the absence of all evidence on physical examination. The *x*-ray lent no support to this view, and it was, therefore, abandoned. Against this theory also was the history of a primary acute infection and repeated recurrences of this infection with chills and fever. On this basis there were considered the possibility of an abscess, possibly with pressure on the bronchus, with perhaps edema of the mucosa, enlarged glands, and especially a gumma. The leukocyte count at no time was over 13,000, even in the presence of chills and high fever.

One more possibility was considered, and that was a bronchiectatic cavity. This was rendered very improbable, first, by the absence of all copious expectoration, and, second, by the absence of confirmatory *x*-ray evidence. It was considered

possible at this time, then, that there was either a gumma or a group of luetic glands pressing on the large bronchus. He was given mild antiluetic treatment at this time.

The following month he had attacks of weakness and dizziness at various times, during which his pulse was irregular and weak and his nails were blue. He had constant wheezing. He slept poorly at night, was very dyspneic on exertion, but had had no precordial pain for almost two years. At this time there were numerous sibilant râles over the left lobe. His pulse was very irregular (extrasystoles).

In October, 1922 he was in bed for two weeks because of dyspnea and constant wheezing, but there was no precordial pain. He had one severe chill on the 12th of this month, with lancinating pains in both legs. Temperature was 103.6° F. The following day his temperature was normal. His daily temperature varied from 99.2° to 98.8° F. Pulse was 64 to 72, with occasional extrasystoles. There were breath sounds present in the left upper lobe, weaker, however, than in the right, almost inaudible over the left lower lobe, and occasional sibilant râles. Fremitus was present over the left upper and diminished over the left lower.

On December 25, 1922 he had been coughing more severely in the last few days and had been nauseated. Numerous sibilant râles were heard on the right side, and no breath sounds at all could be heard over the left.

On January 17, 1923 it was noted that he was still short of breath, had pain on walking, and expectorated slight amounts of blood at times. Two days ago he had a temperature of 102° F., with delirium. Temperature lasted but one day. Expansion was greatly diminished on the left side, numerous râles on the left side and none on the right.

On January 20, 1923 there was impaired resonance over the left lower lobe and weak breath sounds, while over the left upper lobe there was normal fremitus and quite audible breath sounds.

On April 4, 1923 it was noted that he began to have pains in the legs four days ago, with chills, fever, delirium, attacks

of coughing, pallor, and a feeling of constriction in the head, the latter being relieved by nitroglycerin. The fever of 102° F. continued for three days, with sweats on the last day. During this time he had no cough, but next day, after the fever had subsided, his cough began. During the attacks no breath sounds were heard over the left side. The following day he expectorated more profusely, the sputum being whitish green in color, with no odor. On this day breath sounds were again audible over the left upper lobe and almost normal over the left lower. This was interpreted as an increase in pressure on the left bronchus during the attack, with a diminution of pressure most marked over the left lower lobe. This was accompanied by an increase in expectoration, a lowering of the temperature, and an increase in breath sounds. During the whole attack the leukocyte count was normal.

At this time the condition seemed to be progressively getting worse and, therefore, in spite of the cardiac condition, an active antiluetic treatment was instituted. This was carried out by Dr. Erwin Zeisler, twelve intravenous injections of neosalvarsan, 3/10 gram each, being given during June and July. The improvement during this time was quite marked. The dyspnea on exertion was less marked, but the wheezing and the nightly autothony remained. The febrile attacks, however, diminished markedly in number and intensity. A number of *x*-ray examinations were made, but without definite result.

January 4, 1924 it was noted that the patient had a second course of neosalvarsan, and felt quite well until a few days ago, when he again had pains in his legs and a temperature of 101.2° F. Last night had a pain in the precordium, extending down the left arm. There was impaired resonance in the left intrascapular region, with diminished fremitus and weak breath sounds and an occasional dry râle.

On April 23, 1924 the following note was made: In Atlantic City two weeks ago had a severe diarrhea and cough, with bloody expectoration. He returned home and yesterday had severe pains in the legs, with temperature of 102° F., slight delirium, and weakness. Last evening had a sudden attack of severe dyspnea

and felt as though he were about to die. Quite suddenly he coughed unusually hard and expectorated a small, hard, irregular

Fig. 109.—Expectorated calcaneous nodule.

mass with a small amount of blood-stained sputum. His dyspnea ceased instantly and this morning he feels well. There is a marked increase of breath sounds in the left side of the chest,

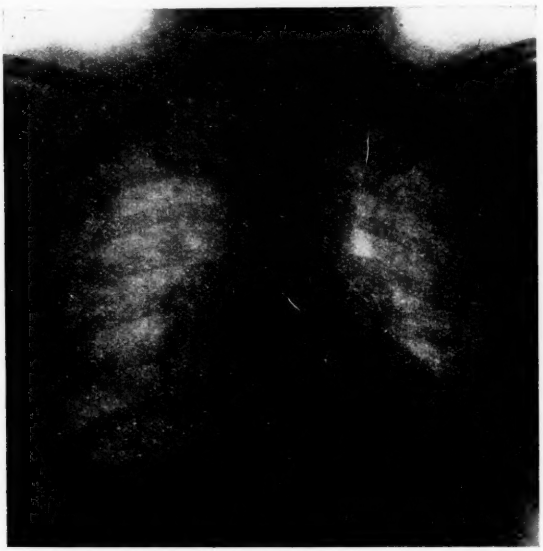


Fig. 110.—Outline of calcified area in left hilum. Note similarity in shape to Fig. 109.

with normal fremitus, a moderate number of medium-sized, moist râles at the left base.

On April 27, 1924 it was noted that he felt quite well, had no wheezing at all, no dyspnea on moderate exertion, no expectoration. The breath sounds on the left side and the fremitus are normal. The heart is as before. Has an occasional crepitant râle at the left base.

On examination of the expectorated nodule, a reproduction of which is given in Fig. 109, it was found to consist of a hard mass, about 4 x 3 x 2 mm. On comparing it with the *x*-ray plates taken at various periods of his illness, we were struck by the similarity of the contour between this and the calcified area in the left lung indicated by the pencil outline reproduced in Fig. 110. We considered this at this time to be simply a calcified gland and paid no further attention to it. In the light of the resemblance to the contour of the expectorated nodule, we considered the possibility that this might be the source of the pneumolith. As seen, it is situated at the level of the seventh rib which is about the origin of the first branch of the left bronchus, and it is probably a result of the acute infection which took place in December, 1921, resulting in a calcification of that gland, or possibly was simply a reinfection of an old calcified gland.

The subsequent attacks of chills and fever evidently resulted in a softening of the matrix in which it was embedded, including the bronchial wall, and a moderate obstruction, especially of the left upper bronchus, was the result. The left lower bronchus was occasionally involved when the inflammatory edema became more marked.

The type of infection cannot be determined. There was no specific organism ever found in the sputum. Whether lues had any part in it we do not know, but there was apparently a decided improvement after the neosalvarsan injections. The patient at present is more comfortable than in many years.

CLINIC OF DR. RALPH C. HAMILL

NORTHWESTERN UNIVERSITY MEDICAL SCHOOL

TICS

THERE are a large number of so-called involuntary movements which need differentiation from each other because of the essentially different sources of origin and mechanism of development. Since the work on the corpus striatum, localizing in this region the origin of certain tremors and even of larger movements, a long step in the understanding of involuntary movements has been made. The number of these involuntary movements which might be considered hysteric has been considerably cut down.

However, there are still certain types of movements which have to be considered as of purely mental rather than physical origin. These movements may all be classed under the heading of "tics," but it is better to stick to the older idea, which presupposes some form of compulsion, some necessity of expression, in the background of the movement. If we attempt to make a definition of a tic we are forced to include the idea that the tic relieves a desire on the part of the individual to perform a certain act. It is for the sake of this relief that the tic is continued. If in this definition we attempt to describe the act we will have to say that any motor act, whether purely muscular in the sense of producing movement or in the sense of producing a noise may become a tic if added to the motion itself there is a sense of compulsion experienced by the ticquer. It is also necessary to include in the definition the idea that whereas in the beginning the movement or tic served some purpose, with constant repetition the original purpose is lost sight of and the relief of tension has taken its place. If we attempt

to shorten up the foregoing remarks into a concise definition, a tic, then, is a movement or an act which originally was for some definite purpose, but which with constant repetition has become habitual and is indulged in as a relief of a sense of tension.

Historic.—Tics were first described as occurring in horses in the middle of the 17th century. The word "tic" may be onomatopoetic, that is, it may represent the noise made by the clicking of the horse's tongue as he cribbed. Perhaps in this day of automobiles but few know what a "cribbing" horse is. A horse can be seen to fasten his teeth in a piece of wood at the side of his stall or at the end of his manger, and with his teeth so fixed take in his breath sharply. As he does this, there is a clicking noise from the tongue. As originally described, it was considered that this activity of the horse was the result of a bad habit. In the middle of the 18th century the term "tic" was applied to man, but it was not until the middle of the 19th century that it was realized that tics were certainly of mental origin. The work of Charcot and his school finally culminated in the book of Meige and Feindel, published in 1902, a classic of its kind.

Etiology.—Tics must be considered as the expression of some defect in personality. This by no means necessarily condemns the personality, as tics are indulged in by men of note as well as by the insane and the feeble-minded. Because there is no definite anatomic knowledge upon which we can base an understanding of personality there is no physical etiologic factor to which we can ascribe the tic. However, the ticquer usually can be shown to have neurotic manifestations in his family tree. His progenitors have been neurotics, have been insane, have been drinkers, inveterate smokers, have been too sensitive, or too high-strung. Tics usually begin between the ages of five and fifteen, or perhaps it would be better to say that some habit which serves no good purpose can be found in the history of those who tic between these ages. For example, a spasmodic cough or, more commonly, finger-nail bitting, thumb-sucking, or twisting of a lock of hair. Any unusual irritation or stimulation from the surface of the body or from any mucous membrane may have caused a movement, a sniff, or a cough for its

relief. Because of the satisfaction gained by the act the act may be repeated. When its repetition becomes frequent and habitual it is to be designated as a tic. Emotional influences, such as embarrassment or self-consciouness, act to increase or continue the bad habit. Under these circumstances one gets the impression that the tic is in the nature of a distraction. Perhaps it is just to say that a tic occurs only in the oversensitive. It is as though such a person was abnormally conscious of sensations from the region of the body involved, and because of this appreciation was forced to respond. Fatigue frequently seems to contribute to the causes of tic. Men and women are equally affected by this disorder. Perhaps one might mention the fact that the ticquer is worse when he has to speak, and many of the tics about the face, head, and neck are observed to be more marked with the beginning of speech. If you will observe two people in a conversation the listener will sit quietly until it is his turn to speak, perhaps this is truer of men than women. At any rate, the listener sits quietly until, just as he starts to speak, he changes his position, starts to scratch his head, picks up something from his desk, coughs in an embarrassed manner, or makes some movement at the same time that he starts to speak. This happens so frequently that it seems as if it must have some reason for its occurrence. Two cases of tic which I have under observation at the present time, both of them movements of the head, always break forth into ticking just before they begin to speak. It seems to me that this tic and the restlessness of the beginning speaker are related. Why does the beginning speaker make his motion or show his restlessness? When a person begins to speak his attention must necessarily be centered on the ideas he is about to advance. There is, however, a secondary attention that is concerned with the effect of what he has to say upon his auditor. This might be called self-consciousness, which, in an exaggerated degree, is embarrassment. Is it not possible to imagine that the act of movement serves to distract his attention from the self-consciousness. The act is the result of cortical innervation. The cortex must be occupied in its production, yet no man consciously says to

himself as he begins to speak to his vis-à-vis, now I will pick up that pencil and tap it on the desk, or now I will shift in my seat. In other words, the act is done with that attention which is not occupied with the ideas and their enunciation. It is as if the motor phenomenon saturates or completely occupies that part of his attention that otherwise might be occupied in self-criticism, leading even to embarrassment. As I have already said, tics occur in the oversensitive. They are oversensitive to their audience. In this sense I believe that tics are fundamentally related to stammering.

Varieties.—Tics may occur in any part of the body. They are, however, most common about the head, face, and neck. Perhaps we should include herein the shoulders. There may be a twitching of the scalp, a movement of the ears, a blinking of the eyes, a lifting of the eyebrows, a grimace of one or both sides of the face, a protruding of the lower jaw, a nodding, twisting, turning, or throwing back of the head, or a shrugging of the shoulders. A cough may become habitual, and in that sense become a tic, or there may be a clucking of the tongue or a sniffing or a grunting. Tics may be quite simple and involve only one or two or three muscles, or they may become elaborated into what seem complicated purposeful acts. For example, a man I know as he begins to talk is very apt to regard his outstretched hands as if to see whether his finger-nails are clean or not. Certain people seem to gain satisfaction in cracking their finger-joints. Tics of the trunk muscles and the legs are not common, yet perhaps the man who studiously avoids stepping on a crack is responding to the same type of mental mechanism as the typical ticquer.

Tics have been divided into tonic and clonic varieties. This is purely arbitrary, since some of the more complicated tics can scarcely be classed as either. However, the commoner forms of tic are usually quick, and hence belong to the clonic variety. The commonest tonic tic is the mental torticollis, which means that the chin is turned over one shoulder or the other and held there for a few moments, to minutes, hours, or all day. This is, perhaps, the most disabling form of tic, and hence deserves special attention. It may start as a mere shaking of the head,

as if one were saying "No," and gradually become more and more exaggerated and prolonged. Of course, it has nothing to do with what is called congenital wry-neck, and, so far as I know, there has been no anatomic basis found for a torticollis coming on as it usually does after the first or second decade. In fact, I think we can say that mental torticollis rarely occurs in the young, but there is frequently a history of some form of tic in the earlier history of the sufferer from mental torticollis.

As I have said, the tic may commence as though the individual were saying "No." This was the case in one woman who found it extremely difficult to say "No" to the solicitations of her men friends. Another case of mental torticollis developed in a man who had to daily walk through a collection of his striking employees to get into his factory. It was as though he could not resist the constant impulse to look behind him. Torticollis has been described as occurring in the nature of an occupational neurosis. That is, in a porter who ordinarily carried his loads on his left shoulder and grew into the habit of turning his face toward that side; or, again, in a copyist who habitually turned his face toward the left where his copy lay. All these causes would not produce a torticollis if the personality defect was not in the background.

Diagnosis.—The diagnosis of tic to one who has seen much of it may be extremely simple. That is, it is obvious that the involuntary movement is a tic. I said involuntary, but I should not use that word in this connection. Habitual is what I meant, but, as I said, it may be obvious that the habitual movement is a tic and is not a manifestation of chorea or jacksonian epilepsy, or any other disease which perhaps undeservedly is considered a more serious thing than the tic. But to recognize the nature of the movement is merely to indicate the line along which the inquiry into the disorder should proceed, namely, the personality side of the case, for, as I have told you, it is the personality defect that constitutes the *sine qua non* of the disorder. Studies of personality are becoming more and more recognized as necessary parts of a diagnosis of any condition that can be called a neurosis, and without a proper valuation of the various facets of a per-

sonality one cannot say that a thorough-going diagnosis in the case of tic has been attempted. I say "attempted" because it is frequently extremely difficult, if not impossible, to hold on to a ticquer long enough to get more than a bare realization that one has to do with an oversensitive individual. These patients are notoriously wanderers through the medical fields. However, in the diagnosis of a tic we have to recognize that it is the frequent, fairly exact repetition of such a movement as might be made at any time by anyone. It is usually sudden, and when first made I presume it might be recognized as studied, or planned, but when habit has made it thoroughly familiar to its owner it often gives the impression of being done somewhat surreptitiously, as if to avoid notice, or when it is in a person with whom we are familiar we may observe that when they meet a new individual, somebody to whom they are perhaps attracted or upon whom they wish to make a good impression, then the tic may be slightly modified, so that it appears to serve some purpose. For example, if it is a blinking tic, the ticquer may rub the corner of his eye just after the movement; or, if it is a shrug of the shoulder, may reach up and put his hand on his shoulder, or rub the side of his neck, or make some other gesture to the effect that there is a discomfort that is causing the action. Frequently it is possible to make the diagnosis at once by merely asking the ticquer to repeat his tic. Some will oblige and some will not. If it is done it is strong evidence of the mental origin and nature. The ticquer practially always can control his tic for a time, then when he relaxes control he will usually indulge in an orgy of tics.

Differential Diagnosis.—In the differential diagnosis tics may have to be distinguished from all other forms of what appear to be involuntary movements. If it is possible to secure a declaration from the ticquer that his disorder or his movement is made for the purpose of relieving a sense of mental tension, the differential diagnosis is accomplished. Such a declaration is not always to be obtained. Perhaps the most frequent condition confused with tic is St. Vitus' dance, or acute infectious chorea. This usually occurs in children, and in children it is extremely

difficult to place much value on response to questions concerning their feeling, as, for example, this mental tension. But when a child is properly approached it is usually easy to get him to tic. It might be said also that a tic is a clean-cut movement that is frequently, and one might almost say exactly, repeated. If a ticquer is shrugging a shoulder that shoulder will be repeatedly shrugged in very much the same way. On the other hand, the movement of chorea is not clean cut. It has been spoken of as amorphous in contrast to the clean-cut movement of tic. A shoulder may be shrugged, but the next involuntary movement may be a twitch of the face, a movement of the trunk, a turn of the head, etc. Acute infectious chorea is, as its name implies, an acute infectious or inflammatory disease with an onset that may be quite definite, though this is not necessarily in the picture. There is, however, some instability of temperature, which may even be a decided rise, perhaps up to 103° F. or more, with malaise and considerable irritability.

Perhaps the most difficult condition to differentiate from tic is spasm. As like as not you will find in most text-books the statement that the reflex origin of the spasm can be discovered and can be used as an aid in the differentiation. In my opinion this statement is somewhat overdrawn. An infected tooth, a sore mouth, an infected ear, a conjunctivitis may be the source of the facial spasm, but very often the removal of the infection has little effect on the spasm. A spasm is a sudden powerful contraction of either a portion of a muscle, the entire muscle, or all the muscles innervated by some single nervous unit, either a spinal segment or a peripheral nerve. A tic is rarely, if ever, on what might be spoken of as a lower nervous basis, that is, a tic is an expression of cortical activity in its beginning, a purposeful act; a spasm is a muscular contraction due to spinal cord or peripheral nerve impulse. Therefore, a tic is not confined to the muscles that are innervated from the lower portion of the central nervous system. Perhaps the most difficult diagnostic question is that of differentiating facial spasm from facial tic. The spasm is always exactly repeated. One might say that it is always of the entire facial musculature on one

side. It is frequently complained of as very distressing or even painful. It cannot be reproduced upon request. A tic, on the other hand, may merely cause a blinking of the eye, an elevation of the eyebrow, or a grimace of one side of the face. It is not as exactly fitted to a pattern as the spasm. There is rarely any sense of discomfort, but rather some of relief. A spasm is practically always unilateral, the tic may be bilateral.

Jacksonian epilepsy may be confused with tic. It is of cortical origin, and the movements may simulate purposeful act. However, this is unusual. The jacksonian attack is always of the same type except that it progresses gradually in the direction of a complete convulsive seizure, whereas the ticquer gives the history of a habit of grunting, or sniffing, or blinking the eyes before what seems to be the convulsive movement of, let us say, the hand appears.

Athetosis may simulate tic. They both are most frequently seen in children. Athetosis is usually shown in connection with voluntary movement, that is, when attempting to grasp an object the fingers and hand assume an exaggerated posture of extension and then contraction, or there may be exaggerated movements of the arm or shoulder or face when these parts of the body are called upon to fit in to some movement pattern, such as the swinging of the arm in walking or, more especially, running, and movements of the face in talking. Athetosis practically always is associated with some change in the deep reflexes, and quite commonly a feeble-mindedness or a history of convulsive attacks is part of the picture.

Treatment.—Tic is merely a symptom. It is an indication of a personality defect. It may be the only indication of defect or the individual may be an imbecile. Naturally an imbecile or the ticking of an imbecile is not to be cured. Imbeciles can be educated up to a certain degree, but beyond that degree "nobody home and nothing doing." Where the personality is functioning fairly successfully then attention may be directed solely to the tic. Such individuals are rather rare. Much the commoner problem is that presented by an individual who tics, but who also complains of embarrassment, shyness, restlessness,

and perhaps difficulties with appetite, sleep, bowel, or sexual function. It then becomes the duty of the doctor to try to get the patient to take a more philosophic attitude of mind toward himself. This may be extremely difficult. One patient may welcome such a procedure, another may object to any inquiry that they consider impertinent. Perhaps the best way to begin treatment with such an individual is to try to get him to understand that the tic is merely the outward manifestation of a lack of proper adjustment within. Then, if that idea can be gotten across, the patient may be open to some kind of suggestion or may, under the guidance of the physician, acquire some better understanding of himself.

In a number of cases tics have begun to be a burden to the patient or the patient has dated their onset with the beginning of the contest with masturbation. In some cases it has seemed helpful to get the patient to understand that the mental mechanism of masturbation and ticking are practically identical. Naturally this exposition has to be approached with proper consideration from the ordinary point of view toward masturbation.

Surgical measures other than perhaps blood-letting are contraindicated. A great many cases of mental torticollis have received some sort of surgical treatment or other. From the foregoing remarks concerning the personality side of this condition I think you can understand how irrational surgical procedures are.

What is true of surgery is true of medicine, with perhaps this reservation. If the ticquer is very nervous and restless 1 or 1½ drams of sodium bromid a day may aid in the acquisition of self-control. As for other medicines, those powerful drugs, valerian and asafetida, powerful to the nose and the taste-buds, may be of some use in children or credulous grown-ups. They know they are getting strong medicine because it tastes strong, and they know they hate it and would do most anything not to have to take it any more, even to the extent of practising some self-control.

Tic may be said to be the result of the lack of proper cortical control, hence a building up of this control is indicated. Since

most of the offenses of tic are unnecessary movements, immobility is to be practised. A régime for its care should include periods of immobility. Also, since we are attempting to increase cortical control of the muscles, exercises are to be prescribed and carefully followed. Since most of the tics are of the head, face, or neck, it is well to do the exercises before a mirror. The following is a general scheme which may be varied to fit the individual case. It is to be repeated three or four times a day:

1. Immobility for ten seconds; each day increase five seconds up to five minutes.
2. Nodding ten times.
3. Throwing the head back ten times.
4. Turning the chin over the left shoulder.
5. Turning the chin over the right shoulder.
6. Immobility for ten seconds; increasing as above to two minutes.
7. A prayer or a piece of poetry should be recited during the first period of immobility.
8. The exercises are to be counted aloud.

During the first week these exercises may be done standing, the second sitting, the third lying, the fourth with the arms outstretched, etc., varying the posture from time to time. It is well at the beginning to see the patient go through his exercises at least once each day. Whenever a tic interrupts one of the above eight divisions the patient must stop and begin that division over again. For example, if it is the immobility, and instead of staying immobile for the prescribed ten seconds, a tic occurs after five seconds, then the period must begin again. If interrupted a second time, that division is passed over and the next taken up; or, if in turning the chin over the shoulder a tic interferes with the smooth movement the second or third time, it should be begun again so that the ten times are made without flaw. It is well to have the patient talk during the exercises, and, furthermore, the talk must mean something to him, not the mere recital by rote of some sing-song affair. I mean that the patient must have his mind on what he is saying. The purpose of this is suggested by what I said about the primary and secondary

attentions. If the primary attention is on what is being said and the secondary attention is occupied with doing some planned remedial exercises, then the secondary attention may be considered to be in the process of education instead of being allowed to run loose.

A very necessary part of the treatment is the frequent encouragement to be given the patient. Of course, this has to run hand-in-hand with the process of finding out the personality defects. In other words, the patient during this process may become extremely discouraged with himself, and if the doctor cannot keep hope alive he had better not begin.

Here are a few cases of ticquers sketched in their bare outlines.

Case I.—B. H. is nine years old. His mother had hyperthyroidism, a thyroidectomy, and two nervous breakdowns. The patient was thought to have something the matter with his throat, because at the age of five he began a nervous hacking, and he stammered somewhat, so a tonsillectomy and a clipping of the palate were performed. Just what their value may have been I do not know. Frankly, I mean that I do not know, since, after all, if the nervous hacking was for the fun occasioned the palate clipping may have been just as logical as spanking the sugar-hungry jam stealer. D. H. was said by his mother to be very sensitive. His stomach was always easily upset. Besides the nervous hacking he has had facial twitchings for the past year.

Case II.—M. Y. is twelve years old, was five and a half weeks premature. She was always considered very babyish and slow to use her hands. Still her mother says that she can play almost anything she hears on the piano. One wonders just what the mother means. M. Y. hacks, twitches her shoulders, grasps her right forefinger between her left thumb and forefinger and rubs it hard, twists the ribbons of her clothes or a lock of hair.

Case III.—I. B., aged sixteen years. Her mother has chronic headaches and had a nervous breakdown when I. B. was four

months old. Even up to the present I. B. wets the bed about every three or four weeks. At eleven she had headaches more or less for a year. This was relieved somewhat by glasses, but a few months later she began jerking and twitching her head and face and shrugging her shoulders. She was kept from school for a few months, improved, and then went back to school, but the twitching returned. She says that she can control the movements, but it makes her restless to do so, and the movements give her some relief.

Case IV.—F. P., twenty-one years old. His father is a nervous type of man. There were no particular bad habits noted in F.'s youth. In fact, it was not until he entered high school that the tics appeared. They are particularly marked of the head and neck, and some of them suggest the nodding movements of the head of some people who wish thereby to emphasize what they have to say. When I was describing my idea of the mental mechanism of embarrassment, F. interrupted to say, "Yes, I remember when these began that I felt that I had to emphasize what I had to say with nodding my head." He also dated the beginning of these tics with the beginning of his conflict with masturbation.

These cases give some idea of the general problem of the ticquer. To work out the personality defect may be impossible or may be merely difficult, but if it could be worked out with more children, I am sure that it would be a good step along the line of preventive medicine; that if these ticquers could be straightened out when their tics first appear in their childhood there would be fewer neurotic adults.

CLINIC OF DR. ARTHUR R. ELLIOTT

ST. LUKE'S HOSPITAL

CARDIAC ANEURYSM

THE subject of aneurysm of the heart possesses not only the element of unusual interest inherent in the rare happenings of practice but also a substantial clinical importance because of its connection with coronary arterial disease

I am fortunately in a position to lay before you the clinical histories and pathologic details of 2 cases of aneurysm of the the left ventricle, one of these cases being of unusual pathologic interest. Before proceeding with the demonstration of this material it will, I think, add to the clarity of our understanding of the subject if we first discuss the pathologic events which precede and eventually result in the formation of aneurysm of the heart wall. It is more than usually necessary that we approach our subject in this manner, since cardiac aneurysm can rarely be recognized during life by any signs or symptoms that are peculiar to it, consequently, aside from coronary disease, upon which it depends, aneurysm of the heart possesses mainly a pathologic interest.

We may look upon the heart as a highly specialized dilatation of the arterial system skilfully and ingeniously adapted for the performance of the part which it plays in circulatory dynamics. It is exposed to the innumerable strains and stresses that fall alike in some degree upon all parts of the vascular system. These it withstands successfully so long as its nutrition is not impaired by disease. Under the operation of degenerative processes the heart wall may yield to pressure just as does the aorta or other artery similarly affected. If the disease process act diffusely, affecting the myocardium equally and in its entirety, the result

will be a more or less general dilatation of the chambers such as is observed in the heart failure of chronic valvular disease or high blood-pressure. If, however, it affects the heart muscle in some circumscribed area leading to local softening and thinning of the myocardium the portion of the heart wall so affected, if it be large enough, may yield to pressure giving rise to parietal bulging constituting the so-called cardiac aneurysm. In this manner we see duplicated in the heart events similar to those so frequently observed in the aorta. The difference between the two is less one of mechanicopathology than of etiology.

Focal weakening of the heart wall sufficient to give rise to bulging, generally speaking, can result from but one cause, and that is occlusion of some important branch of the coronary arteries with infarction of the myocardium. The subject of cardiac aneurysm is consequently inextricably interwoven with coronary thrombosis. For many years coronary thrombosis has been recognized as a cause of more or less sudden death in mature individuals. The clinical events developing from such profound disturbance of the heart's nutrition constitute a fairly clear-cut clinical picture which is recognized by keen observers, so that the correct diagnosis of coronary occlusion is often made during life. In the autopsy room deaths from coronary disease are frequently recognized, the fibrotic patches of infarction being apparent. It is perhaps remarkable that the literature of cardiac aneurysm is so meager considering how common is coronary disease. It receives but passing mention in most of the text-books on diseases of the heart, and in a few no reference to it at all is to be found. Lucke and Rea report that in 12,000 autopsies at the Philadelphia General Hospital and Hospital of the University of Pennsylvania aneurysm of the heart was found only 15 times. In rather striking contrast to this report is the statement of LeCount, that in 34 cases where chronic fibrous myocarditis was the cause of death, 5 of the hearts showed scars of such considerable size that pouching outward (chronic parietal aneurysm) had taken place. To reconcile such wide variation as exists between these two reports perhaps some difference in definition of cardiac aneurysm should be taken into account. In 1908

McElroy could find reported in medical literature but 300 cases of cardiac aneurysm.

The usual site of the aneurysm is at the apex of the heart or in the anterior wall immediately above the apex. In Thurnam's series of collected cases, numbering 66, 27 occurred at the apex and 39 elsewhere over the left ventricle. Legg collected 88 additional cases, in 57 of which the aneurysmal bulging was situated at the apex. Hall still later collected from the literature 112 more cases, 92 of which were left ventricular at or near the apex. It is clear from these statistics that cardiac aneurysm usually springs from the left ventricle. More rarely it may affect the three other chambers, the ventricular septum, the septum membranacea, the valves, especially the aortic and pulmonary. The relative frequency in the distribution of aneurysm may be judged from Hall's tabulation of 112 cases—left ventricle, 92; right ventricle, 1; left auricle, 2; ventricular septum, 15; auricular septum, 2. This preponderance of location in the left ventricular wall fits in with the fact pointed out by Huchard, and confirmed by all observers, that the left coronary artery, especially its anterior descending branch, is usually the seat of the obliterating thrombosis. The frequency with which this happens is strikingly illustrated by Wearn's pathologic findings. In 16 of 19 cases studied the occlusion occurrred in this coronary branch. As might be expected, infarction develops in the anterior wall of the left ventricle or the interventricular septum, both of which parts are supplied by this vessel.

The consequences and effects of blocking of the coronary arteries depend on the rapidity with which it occurs, the size of the vessel occluded, and to some extent upon the previous state of the myocardium, whether it be healthy or degenerated. The vast majority of cardiac aneurysms are of chronic origin, that is to say, they result from a gradual silting up or narrowing of the lumen of the artery from sclerosis, with final occlusion from thrombus formation. Much more rarely aneurysm may occur in an acute form as a consequence of embolus during the course of ulcerative endocarditis. Such emboli, being infective, result in myocardial abscess with acute bulging and termination

by perforation or rupture. Chronic cases seldom terminate in such a manner, but usually by progressive myocardial failure. Legg found rupture in only 7.7 per cent. of 90 reported cases. Thrombotic occlusion implies roughing of the intima of the artery by sclerotic or atheromatous disease.

Although the coronaries are generally considered to be end arteries, their smaller branches anastomose freely in the structures to which they are distributed, and cross-connections between arterial and venous radicles have been demonstrated. In consequence of this, coronary occlusion, unless the artery affected be a main one, may not prove quickly fatal. Much depends upon the rapidity with which the blockade comes about. If it be of a principal artery or branch, sudden death may follow quickly or instantaneously. If it be of slow formation and quantitatively less prejudicial, expansion of the collateral circulation may suffice to prolong life and allow the heart to carry on for a considerable, even a long, period. Osler remarks that advanced coronary disease may be present without much disturbance of the heart's function, and a man may get on very comfortably with only the main branch of one coronary patent, practically only one-fourth of the whole system. Aside from the rate at which the obliterative process develops, it is likely that the previous state of the heart muscle may influence the outcome, for it is easy to conceive that a myocardium already undermined by disease may more quickly succumb to further reduction of blood-supply than will a relatively healthy one.

The more or less sudden shutting off of the blood-supply as the arterial occlusion develops leads to certain effects in the infarcted area which have been variously designated anemic necrosis, acute softening, or myomalacia cordis. The initial change is a coagulation necrosis and softening forming a weakened area in the heart wall which may yield to pressure and rupture or, much more frequently, undergo fibrous tissue transformation, leaving the heart wall at this point thinned and poorly equipped to withstand strain. It is at the seat of such a scar produced in this way that aneurysm of the heart always has its origin.

Cardiac aneurysm is usually single. In size it may vary

from a walnut to as large as a child's head. In McElroy's case the aneurysm was so large as to erode the chest wall and appear as a pulsating tumor near the apex region. Walter described a cardiac aneurysm almost as large as the heart itself. The opening into the heart's cavity is usually large, but may be small, rendering the aneurysm distinctly saccular. If the opening be large, its cavity may or may not contain clotted blood. On the other hand, if the opening into the ventricle is narrow, a thrombus usually forms which may act as a buffer and so relieve strain on the thin and weakened heart wall. The aneurysmal sac consists of endocardium, myocardium, and both layers of the pericardium, which is usually adherent over its convexity.

As has been indicated in the foregoing briefly reviewed facts regarding the mode of origin and location of cardiac aneurysm, it is clear that coronary arteritis is its one great cause. With hardly an exception that is the invariable factor in the production of aneurysm of the apex. The usual sequence of events is a gradual silting up or stenosis of the artery by disease, to which is superadded thrombus formation leading to obstruction and infarction. Fibrous tissue replacement takes place in the area rendered anemic by the plug, and in the scar so formed parietal bulging develops, with the formation of aneurysm. It is an interesting fact that as the infarcted area softens there results a localized fibrinous pericarditis which is often strictly confined to the boundaries of the underlying infarct. In the cases where this has not been observed it is likely that the infarction was too deeply placed in the heart muscle to produce secondary surface developments. This patch of pericarditis is apparently produced in the same or a similar way to the localized pleurisy over a pulmonary infarct. Its presence has a decided bearing on the clinical recognition of coronary thrombosis, as we shall see later.

Coronary occlusion, resulting as it does from either embolic plugging or thrombosis, occurs most frequently between the age of twenty and thirty and in advanced life. During youth occlusion results from emboli thrown off during endocarditis or, more rarely, may be caused by infective endarteritis. In ad-

vanced life coronary sclerosis is the cause, and, as might be expected, its incidence is preponderantly among mature males. In a total of 208 cases of cardiac aneurysm collected by Hare, 74 per cent. occurred in males and 26 per cent. in females. Factors of strain or disease favoring sclerosis and calcification of the coronaries, such as high blood-pressure, gluttony, generalized arteriosclerosis, tobacco excess, chronic nephritis, play a part in its production. Unlike aneurysm of the aorta, cardiac aneurysm does not often result from syphilis.

Aneurysm of the heart is often a postmortem surprise and may appear to be the solitary lesion in an otherwise healthy organ. In the great majority of cases during the clinical course the usual signs and symptoms of cardiac enfeeblement are present, while angina pectoris is a fairly common symptom. In a certain proportion of cases even these indications have been singularly lacking. According to Hall, antemortem diagnosis was made only once in 76 cases. It is only in the event of the cardiac bulge being sufficiently prominent to appear on Roentgen examination that the aneurysm is likely to be detected. We can hardly hope, therefore, to make a successful diagnosis of the condition during life. The important problem for us is to appreciate the occurrence of coronary occlusion. This we may be successful in doing if we take the pains to familiarize ourselves with the fairly characteristic symptoms and signs which this development gives rise to. The clinical features of coronary obstruction are so well illustrated by the following case that I shall let it serve as a demonstration of that condition.

Case I.—Mr. J. H., hospital No. 173,792; German nationality, occupation bookkeeper, married, and having 2 healthy children. This patient first came under observation in 1919, his given age at that date being fifty-eight years and body weight 175 pounds. He had, greatly to his surprise and concern, been recently rejected for life insurance on account of high blood-pressure. His only discomforts were occasional headaches, slight vertigo, dyspnea on effort, and following unusual exertion he would occasionally experience a sense of substernal pressure and constriction. On

physical examination the heart was found to be enlarged without murmurs, but with the first sound markedly reduplicated. Blood-pressure was 180/115. The urine was normal in quantity and specific gravity and contained neither albumin nor casts. Phenolsulphonephthalein excretion was 70 per cent. for the two-hour test period. Blood Wassermann was negative. Clinical progress has been observed continuously from the date of first coming under observation, and was not in any way noteworthy until September, 1920, when he experienced a period of disturbed cardiac efficiency with decline in blood-pressure to 135/100 following upon severe exertion. At this time a systolic murmur is noted in the record, and this murmur did not subsequently disappear, although cardiac adequacy became gradually restored under rest and digitalis therapy Blood-pressure returned gradually to 170/100. Clinical notations contain no comment of interest until February, 1921, when it is recorded that patient complained of recurrent substernal distress about 5 A. M. This wakened him each morning and persisted well on toward noon, when it would disappear, not to return until the succeeding early morning. Pulse-rate 72, regular rhythm, blood-pressure 175/105. Obstructed nasal breathing appeared to be a factor in the production of this morning distress. In June, 1921 mild stenocardiac attacks developed if he exerted himself soon after meals. At this time he was found to react poorly to effort tests and was given moderate-sized doses of digitalis. From this date onward he was never free from thoracic discomforts for any length of time. During the summer of 1923, although comfortable while sitting or lying, he could not walk a block without resting to relieve his anginal distress. In August, 1923 he experienced his first definite attack of angina pectoris with typical brachial radiation. This yielded promptly to the administration of nitrites. There occurred also palpitations during which the heart beat irregularly. His arteries were much fibrosed, very tense, pulse 84, and blood-pressure 200/120. Aortic dulness was increased in breadth. Attacks of angina recurred at irregular intervals during the succeeding months. On November 2, 1923 the electrocardiogram disclosed a normal mechanism

in all leads, regular rhythm, rate 84, conduction time normal. The urine at this time showed a trace of albumin with a few casts. During the early morning hours of November 17th the patient was awakened by very excruciating thoracic pain accompanied by urgent dyspnea. There was distinct cyanosis, the chest markedly hyperresonant, but free from râles. Nitroglycerin failed utterly to relieve his pain and distress, and $\frac{3}{4}$ grain of morphin hypodermically was required before comfort was restored. A drop of 40 points in systolic blood-pressure was noted; pulse-rate 96, pulse not alternating. From this date and attack a state of chronic angina persisted, the patient's range of efficiency being so restricted as to permit of only the slightest exertion. The liver was swollen and tender, with moist subcrepitant râles ever present at the lower lung borders. The average range of blood-pressure was 140/90, with pulse-rate seldom less than 96. Gallop rhythm developed and persisted. Medullary dyspnea rendered his nights extremely restless and unsatisfactory. Cough developed, and on January 14, 1924 a further fall of blood-pressure to 90/60, with flooding of the lower lobes and rise of temperature to 101° F., appeared to indicate the development of hypostatic pneumonia. There developed soft edema about the ankles. The hypostatic pneumonia which threatened at this juncture did not develop, although basal pulmonary congestion persisted. The heart murmur, previously of blowing quality, now became rougher and louder, resembling a pericardial friction. There was no fluid demonstrable in either chest or abdomen. It was found that morphin was absolutely necessary. If he did not receive it he remained awake and in continuous pain, and his cough and thoracic constriction became markedly aggravated. The pulse range was from 100 to 120, and in the main the rhythm was regular, with occasional short periods of what appeared to be ventricular premature contractions. At times the arhythmia resembled paroxysmal fibrillation. Although the attempt was made several times, we were not successful in securing electrocardiographic tracings of these short breaks in rhythm. On January 24th the electrocardiogram (Fig. 111) disclosed a normal mechanism, rate 105, with depressed

T wave in Lead I, iso-electric T in Lead III, very low amplitude RS in Lead II. At all times the heart showed gallop rhythm. Cardiac pain occurred frequently, but was seldom of such severe character as during the earlier portion of his illness. Under administration of digitalis the blood-pressure gradually increased to 132/86, and later to 154/115, the day before the patient's

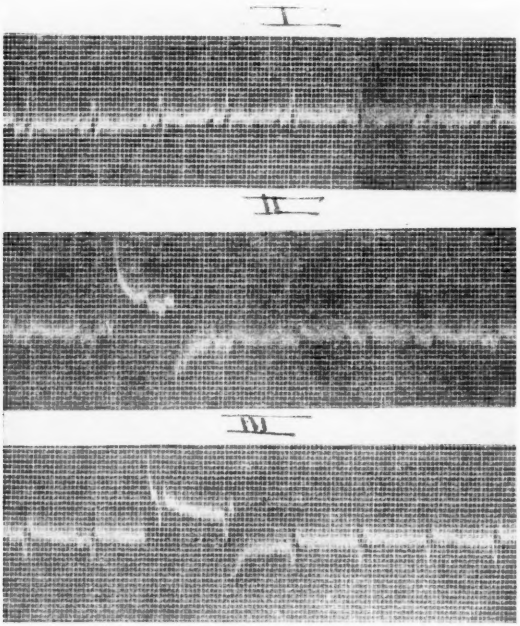


Fig. 111.—Normal mechanism. Depressed T wave in Lead I. Iso-electric T in Lead III. Very low amplitude RS in Lead II. Rate=105.

death. During the night of January 29th there occurred very severe angina requiring ½ grain of morphin. Following this Cheyne-Stokes' breathing developed, edema of buttocks appeared, and the urine volume fell to 500 c.c. From this date progress was steadily downward, angina being practically continuous. Death occurred February 8th from pulmonary edema. The temperature chart displayed an afternoon rise of from 1

to 1½ degrees. The highest leukocyte count observed was 8650. Blood-cultures were negative as late as January 24th, the date of the last one made. Blood chemistry January 16th showed non-protein nitrogen, 35.3 mgm.; blood urea, 13.9 mgm.; blood creatinin, 1.22 mgm. Blood Wassermann negative.

Clinical diagnosis: Coronary thrombosis; pericarditis.

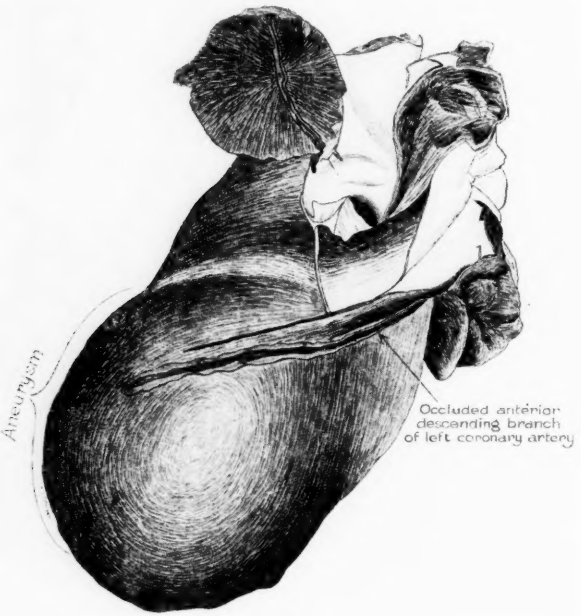


Fig. 112.

I shall now pass around the specimen (Fig. 112) so that you may be able to more clearly visualize the exceedingly interesting description of this heart by our pathologist, Dr. Edwin F. Hirsch:

"The heart *in situ* measures 17.5 cm. in its oblique diameter and 13.5 cm. in transverse diameter near the base. Together with 5 cm. of aorta and 2 cm. of pulmonary artery the heart

weighs 710 gm. On opening the pericardial sac the lower portion is found obliterated by fibrous adhesions between the visceral and parietal pericardium over a region about equal to one-half of the external surface of a baseball. There is a mass of dense fibrous tissue adherent to the apex of the heart, and extending over the anterior and posterior surfaces of the left ventricle covering a surface 9 cm. in maximum diameter. Under this fibrous tissue the heart wall is very thin and soft and bulges beyond the normal heart muscle round about it. There is a network of fine dilated vessels about the periphery of this region extending from the fibrous area on to the heart muscle. There are fibrous tissue changes of the leaflets of the mitral valve and there is a calcified patch 1 cm. in greatest dimension at the attachment of the anterior leaflet, and a slightly smaller one at the attachment of the posterior leaflet. Beginning 4.5 cm. below the attachment of the anterior mitral leaflet on the left ventricular wall there is rough reddish-brown granulation tissue which can be pulled away from smooth endocardium for a distance of 1 cm. Beyond this it is closely adherent to red friable tissue which at that point forms the heart wall. This tissue constitutes the bulging apex of the left ventricle and is continued on the posterior wall and septal wall for 5 cm. above the apex. Above this sac-like bulging the heart wall is dark brown and only 0.4 cm. in thickness and the endocardium above is smooth. The wall of the ventricle increases somewhat to 0.5 cm. at the base. There are fibrous tissue changes of the leaflets of the aortic valve and there are irregularly calcified nodules on each leaflet up to 0.5 cm. in greatest diameter. There are diffuse regions of fatty changes in the root of the aorta. There are fibrous tissue changes of the leaflets of the tricuspid and pulmonic valves. The foramen ovale is closed. The endocardium of the right auricle and ventricle is unchanged. The mouths of the coronary arteries are patent. The right coronary artery is markedly sclerosed, with diffuse calcification for the proximal 13 cm. of its course. Beyond that it is smooth and gray, with only a few scattered regions of fatty change. The proximal 3.5 cm. of the circumflex branch of the left coronary artery is diffusely

sclerosed and calcified, and distal to that it is smooth and gray. The anterior descending branch of the left coronary is markedly thickened and calcified throughout its entire extent, so that it feels like a hard rod on the surface of the heart. At its origin the calcium is so abundant that there is very little lumen for the first 3 cm. of its course. Beyond this point the lumen is distinct for about 5 cm., although the vessel wall is filled with calcium. The distal half of the artery lies along the upper right border of the region of mural softening; and very little lumen can be demonstrated for 6 or 7 cm., it being filled with calcium and firm yellow tissue. The vessel is calcified to its anastomosis with the posterior descending branch of the right coronary artery which appears smooth and gray.

"*Anatomic Diagnosis.*—Myomalacia cordis with saccular aneurysm of the left ventricle, thrombosis of the anterior descending branch of the left coronary artery, marked senile sclerosis with calcification of the coronary arteries, calcification of the aortic and mitral orifices, marked atheromatous senile sclerosis and calcification of the aorta, chronic fibrous mediastinitis.

"*Histology.*—In sections of the heart muscle taken at the junction of the aneurysmal sac and the heart wall, stained hematoxylin and eosin, there is a region about 3 cm. wide consisting largely of dense fibrous tissue staining a very pale blue. This tissue infiltrates the surrounding heart muscle in strands of varying thickness. Many blood-vessels of varying caliber run throughout the substance of this tissue. There is marked fibrous thickening of the arteries.

"*Histopathology.*—Fibrous myocarditis; sclerosis of myocardial arteries."

The interest of this case is considerably enhanced by the fact that we were able to follow the successive developments as they occurred from an early point in the clinical course of the patient's coronary disease. Four years before his death he had high blood-pressure, and it is fair to presume that even then hypertension had already been in existence for a considerable time without his knowledge. He was of the plethoric, obese,

short-necked, physically indolent type so often noted among hypertensive invalids. Already in 1919 he had begun to experience stenocardiac discomforts and marked dyspnea on exertion. The first outstanding incident that occurred under our observation was a period of myocardial insufficiency attended by a drop of 45 mm. in blood-pressure in 1920 following upon severe overexertion. There was no definite angina at this time, and cardiac reserve became satisfactorily restored by rest and digitalis therapy, with a return of blood-pressure to approximately former levels. Although in the interim never free from substernal distress on effort, he experienced his first definite attack of angina pectoris in August, 1923 Recurrence of these attacks during the succeeding months pointed to serious coronary disturbance, yet we find on November 2d, shortly before the beginning of his final illness, that the electrocardiogram was normal in all respects. In particular it is to be noted that there was no T-wave negativity. Just two weeks subsequent to this date a cardiac crisis occurred. During the early morning hours he was awakened by thoracic pain of peculiar and atrocious severity. He affirmed that it was quite unlike previous angina pain and was without brachial radiation. He described the pain as seated deep within the chest and vise-like in its constriction. He sat up struggling for breath, his face suffused and cyanotic The chest was hyperresonant and the breath sounds loud and coarse without râles. The liver came down swollen and tender, the blood-pressure dropped 40 points, and his pulse was thereafter persistently rapid. Nitroglycerin was utterly ineffectual and large doses of morphin were required to relieve his distress. Within a short period gallop rhythm and alternating pulse became apparent. Medullary dyspnea and later typical Cheyne-Stokes' breathing developed. It was obvious that some grave defect had developed in the heart muscle. The clinical picture strongly suggested a coronary thrombosis, and that was the conclusion we arrived at. This attack initiated a status anginosus that persisted with varying severity and without more than transient cessation until death occurred eleven weeks later. As we follow the clinical history we note a steady increase

in myocardial weakness, with the development of pulmonary hypostasis and edema. On January 14th, eight weeks after the first severe cardiac crisis, another critical development occurred characterized by a profound drop of systolic pressure to 90 mm. and great cardiac depression. By this time we were able to detect a friction murmur at the base of the heart, and this was taken to indicate a localized pericarditis over the area of infarction. The electrocardiogram now revealed marked T-wave negativity in Lead I and flattening out in Lead II.

This clinical summary includes practically all the signs and symptoms of coronary occlusion. There is no other development in clinical cardiology that introduces so grave a complex of disturbances. It is, of course, well known that coronary arterial disease may develop to the point of occlusion quite silently, to be revealed only at autopsy. Many cases pursue a stormy course with pretty clear-cut syndromic manifestations which enable the attentive and informed observer to recognize what is happening. The principal symptoms marking this state are pain, myocardial weakness, pericardial friction, and electrocardiographic anomalies.

Pain is sudden and severe. It usually is thoracic, with or without brachial radiation. It may in certain instances be located in the upper abdomen or lower costal region and be associated with vomiting and meteorism, so that it sufficiently resembles visceral perforation as to give rise to some considerable difficulty in interpretation. Patients who have previously been subject to angina seizures recognize at once that something different from usual has occurred. They insist that the pain is less sharp, but more severe and grinding, that it is more vise-like in its constriction, and marked by a not hitherto experienced distress of breathing. In fact, it is different from paroxysmal angina pectoris not only in its quality and severity, but in its duration. It is not amenable to nitrites, and even after morphin it may persist for hours, days, or, as in the case described, for weeks as a status anginosus. This chronic persistent angina raises a strong presumption of coronary occlusion.

With the advent of coronary block grave cardiac weakness

becomes apparent. If the blood-pressure has previously been high, there develops a more or less critical fall in systolic reading. The pulse becomes persistently rapid and may be irregular or alternating. Gallop rhythm is heard over the heart and usually persists till death. Erection of the lung takes place, the chest becoming hyperresonant, as in emphysema, with or without râles. Pulmonary edema may follow. Cheyne-Stokes' breathing is not uncommon. A frequent hacking cough may add to the patient's distress. These developments signifying reduced ventricular power are not, of course, characteristic phenomena, as they may exist without coronary occlusion. A far greater significance attaches to the presence of pericardial friction and to the alterations in the electrocardiogram.

You will observe that on the surface of this heart there is a localized ragged appearance of the pericardium where adhesions have been torn apart covering an area somewhat larger than a doorknob. This represents a patch of fibrinous pericarditis pretty accurately delineated by the boundaries of the underlying infarction and the aneurysm that has developed thereon. We were rather slow in this case in recognizing the signs of pericarditis during life. It is not always easy to catch the friction murmur which may be evanescent, appearing and disappearing. It must be carefully sought for at each examination, otherwise it may easily be missed. Its time of development varies in different cases. It may be heard within a few days after occlusion has occurred or not until many days later. Localized pericarditis is one of the most characteristic signs of myocardial infarction As the anemic area from which the circulation is shut off softens, the overlying pericardium reacts and becomes covered with fibrinous exudate in the same or a similar manner to the formation of localized pleuritis over a pulmonary infarct. Of course, if the area of infarction be on the posterior aspect of the ventricle, no friction murmur may be heard. Gordinier found friction present in 11 of 13 cases, and Gorham, in 5 of 6 cases. The absence of pericardial friction does not invalidate the diagnosis of coronary occlusion. Our examination may have failed to detect it, although present, or the location of the infarct may

be unfavorable to its development. If, however, pericardial friction is detected and there exist rapidly developing grave indications of myocardial insufficiency, especially if there be pain, the diagnosis is rendered practically certain.

I shall not discuss at length the alterations in the electrocardiogram which are interpretable as pointing to grave coronary defects. Those of you who would like to inform yourselves in the matter are referred to the work of Pardee, Herrick and Smith, Kahn, and Willius and Brown. I shall not quote further than to cite from the findings of Willius and Brown,[1] since theirs is the latest publication on the subject. In 48 per cent. of cases of coronary sclerosis studied in which electrocardiograph examination was made, significant T-wave negativity in isolated and combined derivations was noted. In 75 per cent. of these the T-wave negativity was in Lead I only. These authors consider that this anomaly is significant and should always direct attention to disease of the coronary arteris. In our case T wave was negative in Lead I and was iso-electric in Lead II. The autopsy findings confirm the implication.

It is unfortunate that Roentgen plates of this patient's heart were not taken. It is possible that had it been done an antemortem diagnosis might have been made, although the location of the aneurysm renders this very doubtful.

I shall now invite your attention to our second case. This patient's history and pathology constitute an excellent foil to the instance we have just been studying, and the contrast between the 2 cases serves very well to illustrate the wide clinical variation that may exist, although the morbid end-product is essentially the same.

Case II.—Hospital No. 175,165. Male, aged sixty-two; occupation carpenter, admitted March 21, 1924 complaining of dyspnea, orthopnea, and edema of feet and legs. Examining room diagnosis: Cardiac decompensation. The following items of major interest are extracted from the clinical history:

General health had always been excellent, enabling the

[1] Amer. Jour. Med. Sci., vol. 168, p. 163.

patient to follow his trade without interruption or noticeable diminution of endurance until November, 1923. There had been no notable loss or gain in body weight. All venereal infection and intemperance denied by patient. During November, 1923 there occurred rather severe nocturnal paroxysms of non-productive cough. There was a marked decline in general well-being. Within a fortnight dyspnea was continuous and work impossible. Coincidently edema of the ankles developed and the urine became dark colored and scanty. Appetite declined and loss of weight was progressive to the extent of 20 pounds, despite his edema. Aside from a sense of pressure and constriction there had been no pain or other thoracic discomforts.

On physical examination patient was seen to be markedly orthopneic, sitting upright in bed, breathing rapidly and with difficulty. There was decided cyanosis, with marked engorgement of the superficial veins. There was edema of extremities and sacral region. The palpable arteries were fibrotic and tortuous, pulse 112, regular, not alternating; blood-pressure 135/110. The chest displayed the contour of emphysema, breathing being mainly abdominal. Percussion disclosed a flat note below the level of the third rib anteriorly and below the fifth rib posteriorly on both sides. Breath sounds were tubular in quality above the level of flatness, becoming vague and obscure below that line. Voice transmission and fremitus markedly damped over dull areas. Many large moist râles were heard over resonant portions of chest. It was found impossible to outline the heart's diameters, owing to the fluid present in the chest. The heart tones were indistinct and distant. The abdomen was moderately tympanitic. No satisfactory evidence of free fluid in the abdomen could be elicited. The liver margin could be felt—firm, smooth, and tender—5 cm. below the costal margin. Examination of blood and urine disclosed nothing of importance. The electrocardiogram (Fig. 113) revealed a normal mechanism with slight widening of the RS interval, aberrant and low amplitude in Lead II, T wave almost iso-electric in Lead I, low T wave in Leads II and III.

The physical findings appeared to indicate a cardiac break-

down subsequent to hypertensive cardiovascular degeneration of long standing. In view of the patient's severe respiratory distress paracentesis of the left pleural cavity was done, with withdrawal of 1380 c.c. of blood-stained fluid which had a specific gravity of 1013, cell count 463 per cmm., of which 90 per cent. were lymphocytes. On direct smear (Gram) no bacteria were

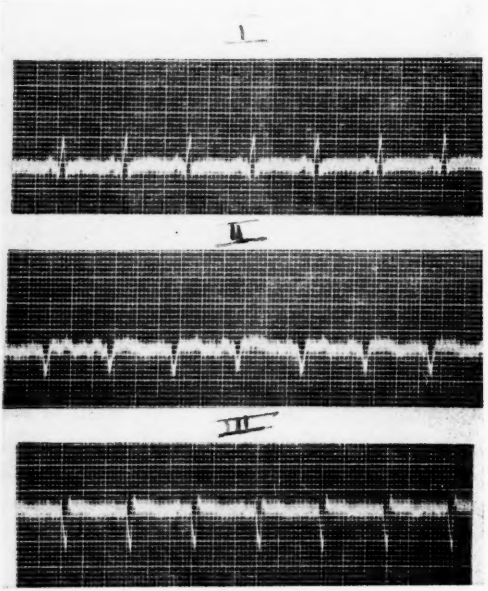


Fig. 113.—Normal mechanism. Slight widening of RS interval, aberrant and low amplitude in Lead II. T wave almost iso-electric in Lead I. Low T wave in Leads II and III.

seen and cultures were likewise negative. No acid-fast bacilli were found. Sputum examinations were also reported negative for tubercle bacilli. A 2-meter *x*-ray plate of the chest (Fig. 114) made on the day following left pleural drainage yielded the following report: "The chest seems retracted on the right. The diaphragm is obliterated on both sides, probably due to fluid in both pleural sacs. The costophrenic angles are obliter-

ated. The heart and aorta are apparently displaced to the left. There seems to be a large well-circumscribed mass located in the right base. The interlobar pleura on the right is thickened. There is probably a localized collection of fluid which extends outward and downard from the posterior mediastinum on the right side accompanied by some fluid in the left base."

Following withdrawal of fluid from the left pleural cavity orthopnea was much relieved. On March 28th puncture of the

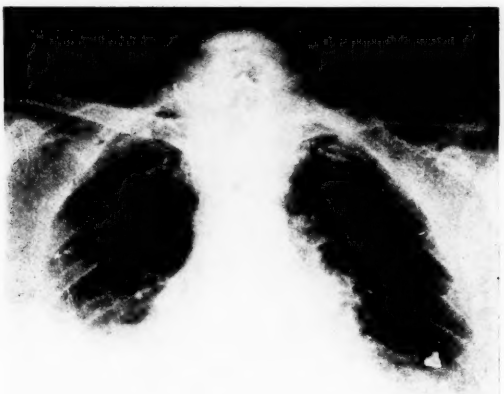


Fig. 114.—Roentgen chest plate. Case II.

right pleura withdrew 930 c.c. of blood-stained fluid which yielded cytologic and bacteriologic findings similar to that previously withdrawn from the left pleura. At this juncture another *x*-ray plate of the chest disclosed a dense shadow practically obliterating the posterior mediastinum. This mass was not of homogeneous character, as there were areas of increased density and areas of increased radiobility. It had more the appearance of a con-

solidation than a tumor or fluid collection. There were no anomalies of cardiac outline. On April 1st the left chest was tapped and 660 c.c. of blood-stained fluid removed. The patient's comfort was now much greater, but his general condition remained otherwise essentially unchanged except for periods of mild delirium. On April 6th the patient was found dead in bed during the early morning hours.

Abbreviated Necropsy Report.—Omitting all unessential details, the following description of the condition of the heart and thoracic organs is extracted from the elaborate report of our pathologist, Dr. Edwin F. Hirsch: The right pleural cavity is completely obliterated by easily broken fibrous tissue. There are also extensive adhesions of the left pleural surfaces. In each pleural sac there is a little fluid between the fibrous bands, the physical properties of which are not certain because of the blood escaping on tearing the tissue. When the sternum is removed its under surface is found to be densely adherent to the pericardium. The pericardial space is very largely obliterated by dark red, easily torn fibrous and fibrinous adhesions, and in the pericardial sac there is about 200 c.c. of blood-stained limpid fluid. The margins of both lungs are adherent to the outside of the pericardial sac. There is partial obliteration of the pericardial space, chiefly in the lower portion, and the fluid is confined more to the neighborhood of the auricular appendages. The lining of the aorta is found to be roughened by scattered yellowish-white elevations 3 to 4 to 15 mm. in surface dimensions and with sloping margins. There is very little calcification of these regions. There are no changes demonstrable in the pulmonary arteries or in the great veins.

The adhesions between the pericardium and heart are densest and firmest opposite the apex and on the left side. When these adhesions are broken and the apex exposed there is revealed a saccular, almost round, projection of the front and lateral wall of the left ventricle near the apex with the heart in position, 10 x 8 cm. in diameter. This projection is covered by white fibrous tissue and reddened granulation tissue. The outside of the heart, especially the auricular portion, is roughened by

masses of fibrin and organizing fibrinous tissue. There are fibrous tissue changes of the leaflets of the pulmonic semilunar valve. The mitral valve shows no gross pathologic change and admits the tips of four fingers loosely. The tricuspid valve admits five finger-tips. The aortic valve is competent to the water test and shows no gross changes. On surfaces made by section of both lungs there is abundant thin, slightly blood-stained fluid.

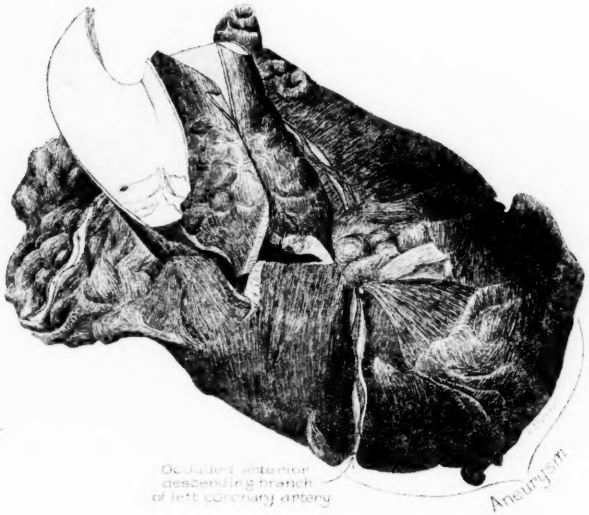


Fig. 115.

When the emptied heart with the thoracic and abdominal aorta and pulmonary artery were removed the combined weight was 742 gm. The surface of the heart is shaggy with fibrous tissue and fibrin, the latter chiefly at the base. There is an almost round ball-like projection near the apex of the heart 10 x 8 cm. in diameter. It extends upward and to the right, displacing the anterior longitudinal sulcus somewhat, so that it forms a curve with its convexity up and to the right. This region is covered with very adherent, dense reddish-gray fibrous tissue. The

coronary arteries are tortuous and their lining roughened by firm yellow elevated patches scattered throughout their extent. At about the middle of the right upper margin of the projection described above and 8 cm. from the origin of the left coronary artery the anterior descending branch of this vessel is firm and cord-like and contains an organizing yellowish-white thrombus. The thrombus is 3 cm. long and the lumen of the vessel has been partially re-established. Beyond the thrombus the artery is patent and it anastomoses with the posterior descending branch. The endocardium of the right ventricle is everywhere reddish brown, smooth, moist, and glistening. The endocardial lining of the right and left auricles is everywhere slate gray, smooth, moist, and glistening. The right ventricular cavity is somewhat distorted by the mass below it; 4 cm. below the attachment of the anterior mitral leaflet there is a smooth white band traversing the septal wall of the left ventricle transversely 1 cm. wide. Above this band the endocardium is reddish brown, smooth and glistening, and below it is a cavity forming the projection at the apex of the heart. The lining of this cavity is roughly granular and reddish gray. It extends upward about 3 cm. on to the posterior wall of the left ventricle, where the granular lining covers the bases of the papillary muscles and chordæ tendineæ, but here there is no distinct line of demarcation as on the septal wall. The cavity is 5 x 8 cm. in diameter, and its walls are about the thickness of the ventricular wall except in front, where it is about half as thick. The heart muscle histology showed changes typical of chronic fibrous myocarditis.

Postmortem cultures from heart's blood and pericardial fluid yielded Streptococcus hemolyticus.

Anatomic Diagnosis.—Chronic fibrous pleuritis; chronic fibrous mediastinitis; chronic fibrous pericarditis; atheromatous senile sclerosis of aorta; senile sclerosis of coronary arteries, thrombosis of the anterior descending branch of left coronary artery; aneurysm of left ventricle near apex.

Histopathology.—Chronic fibrous myocarditis.

As we review the clinical history of this patient we find a fairly sharp and well-defined point of departure from which to

date his coronary occlusion. There is an entire absence of thoracic or abdominal pain both in his previous experience and at or after the beginning of his last illness. It is interesting to note, however, that his disability was initiated by developments which in their abruptness and effects resemble a cardiac crisis. In November, 1923, while enjoying his usual health and daily engaged in his occupation, he experienced a severe nocturnal paroxysm of non-productive cough, with dyspnea, but without pain. Dating sharply from this attack his strength declined so rapidly that within a fortnight he was completely disabled. Edema developed almost from the start. Thereafter the progress of his case conformed to the type of progressive heart failure. If we are correct in dating back the occurrence of coronary thrombosis to his primary discomforts on November 17th, we shall have to allow a period somewhat less than six months during which life continued with a crippled coronary circulation until death (April 6th following). Examination of the thrombosed portion of the vessel disclosed a partial re-establishment of its channel. The circulation via this vessel together with collateral assistance through anastomosis was apparently enough to permit the heart to carry on for this period, but was evidently not sufficient to enable the heart wall to successfully withstand pressure. There was, however, less marked thinning of the ventricular wall at the aneurysmal bulge than in our first case. The extensive inflammatory involvement within the chest cavities and mediastinum, with exudate in pleural and pericardial sacs, prevented detection of pericardial friction. The electrocardiogram disclosed suggestive evidence of grave disturbance within the myocardium, but we failed to give this due weight, and were not impressed with the possibility of coronary occlusion. Autopsy revealed extensive serofibrinous involvement of pleuræ and pericardium. The presence of Streptococcus hemolyticus in the heart's blood and pericardial exudate we regard as a terminal invasion in view of negative blood-cultures during life, and failure to secure bacterial growth from fluid withdrawn from the chest by paracentesis.

Four types of cases of coronary artery occlusion have been described by Herrick:

1. Cases where death is very sudden or instantaneous. If preceded by pain, death is usually attributed to angina pectoris. Autopsy discloses the true nature of the lesion.

2. Cases which are preceded by critical developments and develop physical signs of coronary artery occlusion, death occurring in a few hours or days and usually suddenly.

3. Cases in which death is due to myocardial failure weeks or months after the characteristic abrupt and often stormy onset.

4. Cases with abrupt onset and with the clinical manifestations of coronary involvement which eventually recover and may survive for months or years with crippled myocardial efficiency.

It is likely that the third group of this clinical classification contributes practically all the examples of aneurysm of the heart wall that have been observed. It is to this group that the two instances we have been studying today belong.

CLINIC OF DR. JACOB MEYER

MICHAEL REESE HOSPITAL

SUBPHRENIC ABSCESS—SUPPURATIVE PYLEPHLEBITIS SECONDARY TO A PERFORATING GASTRIC ULCER

I WISH to present this case to you to illustrate (1) the difficulties of bedside diagnosis; (2) the importance of analysis of details in history of physical examination, and (3) positive value of negative laboratory findings.

You must then first imagine yourselves at the home of Mrs. S., past fifty-two years, whom you see for the first time. From her son and her you obtain the rather meager story, that about four weeks prior to your present visit the patient was suddenly seized with severe abdominal pain, located in the epigastric region and also in the right upper quadrant. This day had been a holiday and the patient had "fasted"—and she ascribed her attack to her weakness from fasting. However, the pain was so severe and the general condition so poor that her son asked a local physician to see her. This doctor told him that the patient was suffering with "acute gall-stones" and should be operated, but, as the patient would not consent, he prescribed some medicine which she has been taking ever since the onset. At first she felt better, but in the past week the children have noticed that she is getting weaker, that she sweats a great deal, and that she complains of pain in the right side. Further questions elicit that the patient has been ailing, but has not had any definite attacks of pain; that she has never vomited; that at the onset four weeks ago there was no vomiting or constipation. Your question as to previous history is of negative value.

You now proceed to physical examination, and observe a woman past middle age, who looks rather sick—more so than the

physical findings suggest. The pulse is 90; the temperature 98° F.; the respiration 28. The head and neck are negative. The lungs are negative. There is a tympanitic area in the region normally occupied by the liver. The abdomen is soft and there is no rigidity or evidence of fluid in the peritoneum. Tenderness is present beneath the right costal arch and the liver can be palpated. Otherwise the findings are negative.

As we are still in the patient's home and have no means to obtain laboratory data, we will try to analyze this case and render an opinion before leaving the home. This meager history at once suggests the "*acute abdomen.*" True enough, we have no story of vomiting, but, if you will recall, this patient had been fasting all day, and so it is very likely that this may explain the absence of same. Of the causes of acute abdomen we can rule out:

Appendicitis, because of the absence of any local tenderness in the region of the appendix; because an acute appendicitis of four weeks' duration would be accompanied by a diffuse peritonitis.

Gall-stones.—We may rule these out because of the absence of previous attacks; the absence of jaundice; the local tenderness and rigidity in right upper quadrant strongly suggests this, but we would expect some elevation of temperature. We might go through and mention many other causes of acute abdomen, but in order to save time I will suggest a perforated viscus—either stomach or intestines. This you believe is out of the question because we would expect a peritonitis with free fluid in abdomen and the classical symptoms of ileus, such as abdominal pain, vomiting, tympanitis, and constipation, which are not present.

However, may we say that a rupture of the viscus has occurred in the lesser peritoneal cavity? My reasons for suggesting this are: You remember that physical examination showed a tympanitic note in the region normally occupied by the liver. This means that air has entered the abdominal cavity. Where can the air come from? It cannot be the lungs, for the breath sounds and lung resonance on the right side are normal. It

must enter from some other source, and most likely from the perforated viscus. Again, if perforation occurred into the greater peritoneal cavity, evidence of peritonitis would be present. May we not assume that perforation in the lesser peritoneal cavity may have occurred? I suggest, therefore, a perforated lesion of the stomach, very likely an ulcer, and I say ulcer and stomach, (1) because it is the most likely lesion, (2) because

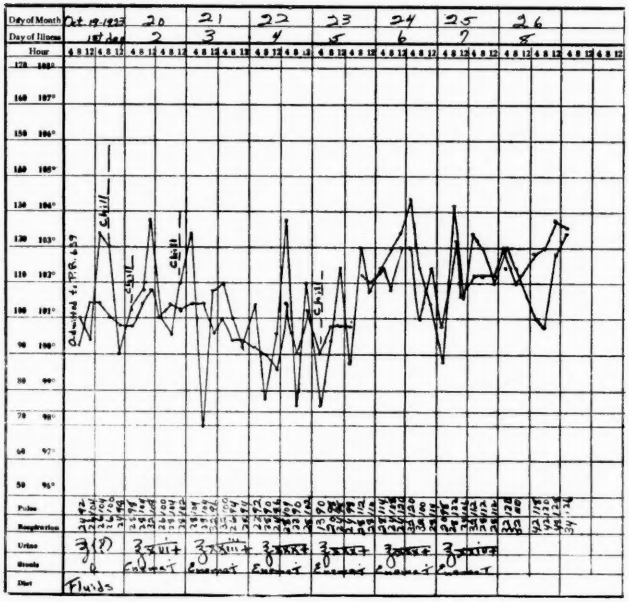


Chart I.

the pain was located in the epigastrium, and (3) because the intestinal tract is free from evidence of ileus.

Let us now see the patient at the hospital the very next day. If you look at the nurse's chart (Chart I) you will note the temperature is 103° F., the pulse 100, respiration 32, and there is a statement that the patient has had a severe chill lasting about fifteen minutes. This is the first time we have mentioned chill or fever, and we must return to our story and see whether or not

this was previously present. The patient is certain that this is the first time she has had a chill or has been very feverish. The physical examination shows no change. Tympany is still present in the region normally occupied by the liver.

May we not have pus beneath the diaphragm? If pus were present the liver dulness would be higher than normal and the breath sounds absent. Further, we may resort to the fluoroscope and *x*-ray, and these, as you observe, show a freely movable right diaphragm and no shadow between the liver and diaphragm. We have even checked this by introducing a trocar, and have failed to obtain pus. The leukocyte count is first 17,000 and has increased to 21,000. The blood-cultures are negative. A blood-smear is negative and studies for the plasmodium are negative. So you see, even though we have used all the laboratory facilities, we are not helped in our diagnosis.

Please refer again with me to this temperature chart, and you recognize that it is a typical temperature curve of *sepsis*. Now the laboratory studies help you to say that the sepsis is not of bacterial origin nor due to the plasmodium.

Clinically we must think of a suppurative cholangeitis or a suppurative cholelithiasis. We are inclined to rule these out because of the absence of jaundice and the absence of any bile-pigments in the urine.

We again suggest *pus* in the subphrenic space, but, as we cannot locate this, we must think of the final possibility, viz., suppurative pylephlebitis secondary to the perforated ulcer, and this would fit well with the story, the chill, fever, sweats, increased white cells, and the absence of any free pus.

Summarizing Briefly.—We are dealing with a patient past middle age who is suddenly taken with severe pain in the epigastrium, after which an interval of four weeks elapses. Suddenly chills, fever, and sweats appear. We say that this picture suggests a gastric ulcer perforating into the lesser peritoneal cavity, and that subsequent to this perforation, either an abscess (which we cannot locate) has formed between the liver and diaphragm, or that a suppurative pylephlebitis has occurred.

After a period of five days, during which the case was care-

fully studied, the patient was explored by Dr. Greensfelder, and I shall read you what was found: "Upon opening the peritoneal cavity a very small amount of peritoneal fluid was present. Upon pulling up the stomach, adhesions were found between the liver and stomach. On freeing these adhesions there was a sudden escape of foul gas and pus into the area of operation, and on the posterior wall of the stomach a small perforation of the stomach was found.

CLINIC OF DR. IRVING F. STEIN

MICHAEL REESE HOSPITAL

STERILITY

I DESIRE to demonstrate today a few cases of sterility to bring out the value of the newer methods of diagnosis and to discuss briefly the prognosis of this condition. Sterility has been classified in various ways, but for our purpose the simple classification of congenital and acquired will suffice.

In the first case we have a woman who twelve years ago gave birth to a baby, and whose husband then immigrated to this country, leaving her for ten years. She followed two years ago, and for the past two years she has been living here, and is desirous of becoming pregnant, but thus far has been unsuccessful. Physical examination does not reveal any prohibitive condition—the genital status is normal to palpation and inspection. There is no endocervical infection and the menstrual history is normal. A condom specimen from the husband reveals numerous live spermatozoa. We then must look for some other source of trouble.

Rubin Test.—One of the most valuable measures at our command is the Rubin test. This test consists in establishing the patency of the fallopian tubes by passing gas or air through the tubes via the uterus. It is a simple procedure, without danger to the patient and causes a negligible amount of discomfort. We shall employ the Rubin test in this case. All that is needed is a tank of carbon-dioxid gas, with a control gage, a manometer, and a water bottle of the Rubin type for measuring a given quantity of gas; sufficient tubing to reach from the water bottle to the table and a modified Ultzmann cannula for introducing the gas into the uterus.

Technic.—Patient in lithotomy position on the *x*-ray table. A bivalve speculum is introduced and the cervix and vagina are painted with tincture of iodin. The anterior lip of the cervix is grasped with a volsellum forceps and the uterine cavity is tested with the uterine sound for length and direction. The Ultzmann cannula is then inserted after testing it with gas and held firmly against cervix. The gas is now turned on very slowly, and while the assistant counts the oscillations of gas in the water bottle, the operator watches the manometer to see whether the gas flows under pressure. If you will take your stethoscope and place the bell on either side of the patient's abdomen just above the pubes you may hear the gas bubbling through the fallopian tubes; yes, here the gas is passing through readily into the peritoneal cavity. Now this gives us a really valuable bit of information. If there is no obstruction to the passage of gas through the tubes, undoubtedly, the spermatozoa can also enter the tubes, providing they are healthy to start with and nothing harms them on the way. Now, this is sufficient for the test alone, but if we wish to go a step farther, we may continue to inflate the abdomen until sufficient distention is obtained to silhouette the pelvic viscera on a film. We then have black on white evidence of the status of the internal genitalia. Any abnormality that escapes palpation will be depicted in the films. By introducing a liter of CO_2 we now have enough distention for a roentgenogram, and, as you see in Fig. 116, a normal genital status obtains.

Why, then, does this woman not conceive? Is she sterile? These questions naturally come to mind as the gradual elimination of the various possibilities for sterility proceeds. This is a case of relative sterility which is probably temporary. In the course of the next few months, without any treatment other than the distention of the fallopian tubes by the gas, this patient will likely conceive. If she does not, we shall do Huhner's test to ascertain whether the spermatozoa reach the cervical canal. We shall report to you later about her.

The next patient I wish to show you is a woman thirty-five years of age, who has been married eleven years and has never

been pregnant. When she first consulted me in 1919 I found that she had a small firm uterus and also that the condom examination revealed only non-motile spermatozoa. The husband was referred to a urologist, who after some months' treatment reported great improvement in his condition and the presence of numerous motile sperms. In July, 1920 the patient again presented herself at my office, with the history that her menstrual period was thirteen days overdue, her breasts were sen-

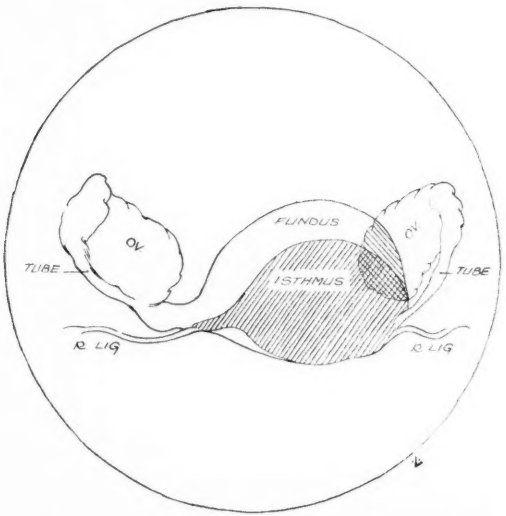


Fig. 116.—Normal genital status. Patent tubes.

sitive, and that she suffered increased frequency of urination. She had never missed a period before and she felt sure that she was pregnant. Upon examination the uterus did not appear enlarged, but a soft tender swelling was palpable in the left ovary. A week later she flowed with clots, and at the end of another week the swelling in the ovary had disappeared. (A corpus luteum cyst would explain this system complex.) Her history was then uneventful until December, 1921, when she missed two periods, and again had a left-sided ovarian cystic

swelling. Meanwhile she was treated with glandular preparations and had a dilatation and curettage, but no pregnancy occurred. She then made the rounds of a number of physicians' offices and received a variety of opinions, and was advised by two physicians to be operated for pus-tubes. She returns now to inquire whether she requires an operation or whether there is anything else to be done.

We can do two things for her. First, we can establish the fact of patency of her fallopian tubes; second, we can obtain

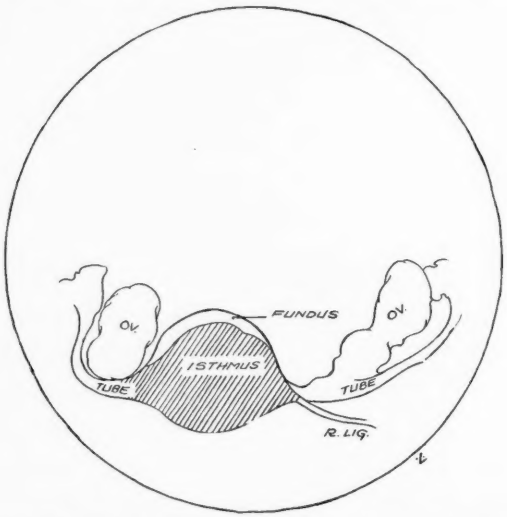


Fig. 117.—Hypoplastic uterus. Patent fallopian tubes. Sterility.

an accurate picture of the uterus and adnexa and thereby conclude whether or not treatment is of avail. (Patency test is made.) You may listen here in the lower abdomen and you will hear how readily the gas bubbles through the tubes; the manometer reads 60 mm. Hear the gurgling on both sides? That disproves the presence of pus-tubes. (The gas was permitted to flow until 1 liter was introduced into the peritoneal cavity, and stereoroentgenograms were taken in the position described by Peterson and Cron.)

The roentgenogram (Fig. 117) shows clearly that we are dealing with a hypoplastic uterus. The shadow of the uterus is not only much smaller than normal, but is uniformly dense throughout and circular in outline instead of oval. The absence of any adnexal pathology is also strikingly portrayed in this film. This, then, is a case of congenital sterility, and the prognosis for cure is essentially bad. There is little hope of increasing fecundity in this type by any method known today.

The third case is a woman twenty-nine years of age who has been married two and a half years. Now, we usually do not consider a woman sterile until three years of normal married life has elapsed without conception occurring (Kisch). However, this woman is anxious for children, and as her husband is considerably older than she, her impatience is somewhat warranted. The physician who sends her to us for diagnosis has assured me that the cervical smear is normal, that libido is normal, and also that the general health of both are good. He has failed to find a cause for her failure to conceive. We find on examination a plump, healthy looking, young woman. The pelvic examination reveals a uterus which is apparently normal in size, position, and mobility. The left adnexa are negative, the right, however, are somewhat tender, and there is a slight enlargement which seems to be confined to the ovary. Smears from the cervix are negative and a leukocyte count is 9000. (Patency test is made.) The gas flows through the tubes readily, with a pressure between 40 and 60 mm., 1 liter of gas is introduced into the peritoneal cavity and stereoroentgenograms made as in the previous case. This film (Fig. 118) shows clearly the pelvic status. The uterus is perfectly normal. The tubes show clearly and are not altered. Both ovaries are somewhat cystic, the left showing a small solitary cyst on the uterine end (may be a corpus luteum) and the right a typical small polycystic ovary. The latter is not of the magnitude demanding surgical interference, but may possibly be a factor interfering with normal ovulation. However, the chances are better for conception if left to nature. This case, as with the first, comes under the heading of a relative sterility and is probably only

temporary. Our experience coincides with that of Peterson and Cron, that pregnancy frequently follows the performance of the Rubin test in such cases as this one and the first one presented.

Just a word in praise of this procedure which we have demonstrated here today. It is a simple, harmless test, and in our clinic has almost entirely displaced the operation of dilatation and curettage, a procedure whose justification and value we

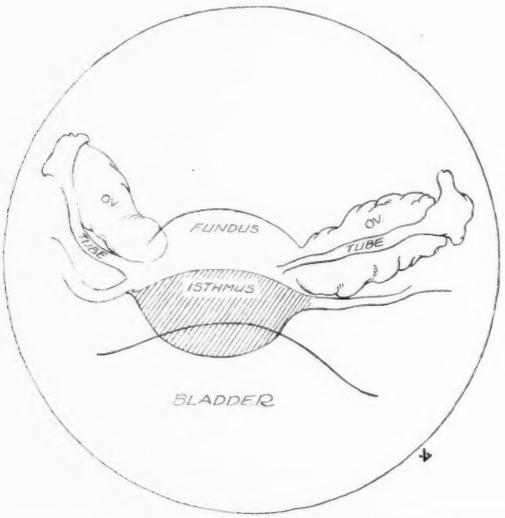


Fig. 118.—Polycystic ovary. Tubes patent. Uterus normal.

long questioned. The use of roentgenograms after producing pneumoperitoneum is also a source of great satisfaction and is highly valuable, especially in settling difference of opinion. Many of our patients have sought opinions elsewhere, as did patient No. 2 here reported, and are in a quandary as to the advice to follow. The roentgenogram after pneumoperitoneum usually leaves no doubt.

Case II is one of primary congenital sterility, and no treatment that we can prescribe will be of benefit. The patient is

not suffering any disease process. She merely has a hypoplastic uterus and is permanently sterile.

Cases I and III are examples of secondary sterility. Patient No. 1 has one child, but now, after an absence of ten years, finds she is unable to conceive. We are unable to detect any pathologic condition she may have acquired since the birth of her child. However, she must be classed as a case of acquired sterility until pregnancy disproves that she is sterile. She may, of course, come under the heading of "one-child sterility."

Typical acquired sterility is that caused by gonorrheal pustubes or by postabortal sepsis. This type is often permanent in nature.

The third case, although never pregnant, is also a secondary sterility, for she has normal genitalia with a complicating cystic change in the ovary. This is usually not a permanent barrier to conception.

(The roentgenograms in these cases were made in the Michael Reese Hospital under the direction of Dr. R. A. Arens.)

CLINIC OF DR. WILLIAM A. BRAMS

MICHAEL REESE HOSPITAL

PLAQUE-LIKE ADENOMA OF THE STOMACH, ITS DIFFERENTIAL DIAGNOSIS AND TREATMENT

THE case which we have for presentation this morning is one of benign tumor of the stomach, which although somewhat of a rarity, is of great interest because it illustrates several important points in the differential diagnosis of gastric tumor and because the proper treatment resulted in a permanent cure.

The patient, a married woman aged thirty-eight, entered the hospital complaining of chronic diarrhea, marked loss of weight, weakness, anorexia, vomiting of small quantities of food and mucus, and vague dyspeptic symptoms, all of which were present for about five years. She had from three to five bowel movements daily and the stools were soft or liquid and contained some mucus, but no blood. There were no other symptoms of importance and there was nothing of any significance in the past history.

Examination showed a very emaciated, middle-aged female who was in poor general condition. Nothing abnormal was found in the chest or abdomen with the usual methods of physical examination. The stomach contents after a test-breakfast showed complete absence of free HCl and but a trace of combined acid. There was a distinct, positive reaction for lactic acid in the stomach contents. The stools, after a Schmidt test-diet, showed the presence of considerable gas formation and a very poor digestion of meats. Numerous chemical examinations for blood were made of the stools, but the results were *constantly negative*. *x*-Ray examination showed a large filling defect of the pyloric portion of the stomach involving chiefly the greater

curvature. The lower pole of the stomach was below the umbilicus and contained a moderate quantity of fluid. The contrast meal began to appear in the duodenum in a few minutes after ingestion and the greater curvature showed a corrugated appearance. The same phenomena were present on several examinations, and a tentative diagnosis of extensive and inoperable carcinoma of the stomach was made. The patient was accordingly placed on palliative treatment until further examinations should suggest some other form of treatment.

The long duration of the illness and the apparent slow course of the disease during the period of observation, together with the presence of diarrhea, led us to suspect that the case was not of the ordinary variety, and that further study was required.

The first point to be determined was whether the presence of diarrhea for five years necessarily signified a primary disease of the intestines. This question was of additional importance in view of the definite changes found in the stomach contents and in the stomach contour on *x*-ray examination, while the symptoms pointed more to an involvement of the intestinal tract.

It is a matter of common observation that patients with achylia or anacidity of the stomach contents are very susceptible to attacks of diarrhea which often becomes chronic. The exact reason for this is not known, but the explanation which is usually accepted is that the absence of acid in the stomach interferes with the proper closure of the pyloric sphincter, and this, in turn, permits an early and rapid emptying of the stomach contents into the bowel. Such early emptying can actually be seen with the *x*-ray, and it is a common experience in patients having achylia that nothing is obtained on aspiration of the stomach after an Ewald meal unless it is performed much sooner than an hour after ingestion. The result of this premature emptying of the stomach is that a large quantity of coarse, undigested food is thrown into the bowel, where it sets up an irritation with resulting diarrhea. Some authors also assume that the absence of acid in the stomach interferes in some way with the proper secretion of hormones, and that the pancreatic

juices are also deficient, thus adding to the difficulties which the bowel has to meet in these cases.

The presence of achylia in our case could, therefore, account for the chronic diarrhea without assuming that the primary pathology is located in the intestines. Such a view would better correlate the findings on *x*-ray examination with the intestinal symptoms and the findings in the stomach contents. The conclusion which was finally reached was, therefore, that the chief lesion was in the stomach and that the intestinal symptoms were complications due to the absence of free HCl from the stomach contents.

The next point to be determined was the nature of the gastric pathology. The marked emaciation, absence of free HCl, the age of the patient, and the filling defect at the pylorus found on *x*-ray examination could all be found in carcinoma of the stomach. This diagnosis could not be accepted without some question in view of the duration of the illness, the absence of signs of metastasis, or appreciable change for the worse during the period of observation which lasted for more than a month. Some evidence of the progress of the malignant disease should certainly have occurred during this time, as the patient was apparently in the last stages of carcinoma, if this condition was at all present. The greatest objection to this diagnosis was the *constant absence of occult blood from the stools on numerous examinations*. Such a constant absence of blood from the stools is very unusual in advanced carcinoma of the stomach, although it is possible to miss the blood occasionally in a series of examinations or if only one or two determinations are made. The presence of blood in the stools in carcinoma is so important a sign that certain authorities, including Boas, state that occult blood in the stools is constantly present in malignancy, while it is present only periodically in ulcer, and that this point may be used in the differential diagnosis between these two conditions. At the same time we must not overlook the early stage of a malignant linitis plastica before ulceration or bleeding has had time to occur. The *x*-ray findings in such conditions are quite distinctive, however, and are not those seen in our case.

Another form of malignant tumor which had to be considered was sarcoma of the stomach. It is very difficult to diagnose this condition, as the subjective symptoms are very much like those in carcinoma. Both sarcoma and carcinoma of the stomach may produce pyloric stenosis, palpable tumor, absence of free HCl, and may produce occult blood in the stools and marked emaciation. Sarcoma of the stomach may, however, sometimes be distinguished by metastasis in the skin, large spleen, or the recovery of tumor tissue in the vomitus or lavage water. The absence of these signs, the great rarity of the disease, and the duration of the symptoms led us to exclude sarcoma as a possibility.

Our attention was now drawn to the possibilities of other conditions which could produce anacidity, emaciation, and a filling defect at the pylorus and which could last for five years without ending fatally. Compression of the pyloric region from without, associated with a marked loss in weight and strength, could produce an anacidity with an apparent filling defect at the pylorus. Such a condition could be excluded by the fact that the *x*-ray findings were constant on numerous occasions in spite of change of posture, and that the outlines of the defect did not change during any of these examinations. No mass was palpable, the liver and spleen were not enlarged, and there was no good ground for supposing that such a compression existed.

Certain chronic inflammatory conditions of the stomach could also produce similar signs and symptoms, and among the most likely are tuberculosis and syphilis of the stomach. Tuberculosis of the stomach is extremely rare, and as the patient showed no evidences of this disease elsewhere in the body in spite of her marked emaciation, the possibility of this condition was hardly to be considered.

Syphilis of the stomach was more difficult to exclude, as this condition may produce achylia, marked emaciation, and similar *x*-ray findings, all of which may last for a number of years. There are several features which speak against such a diagnosis, the chief of which are the absence of luetic findings

elsewhere, negative serologic results, and the failure to respond to specific treatment. The failure to respond to antiluetic treatment is a very serious objection, as it is a matter of common observation that syphilitics not only react well to this treatment but also show a marked tolerance to iodids. Our patient, on the other hand, could not take even small doses of potassium iodid.

The remaining possibilities were limited to a consideration of some non-malignant tumor of the pyloric portion of the stomach. Such a condition, if allowed to persist for a sufficient length of time, could produce achylia, loss of weight, and the peculiar filling defect seen on *x*-ray examination. Such a diagnosis would better correlate the constant absence of occult blood from the stools and the prolonged duration of the symptoms with the other findings. It would be quite impossible to make a differential diagnosis of the variety of the non-malignant tumor which could be present in view of the fact that all types produce signs and symptoms which closely resemble one another. It is only by recovering a particle of the tumor tissue in the lavage water or vomitus that such a diagnosis could be made. Polyps produce a characteristic picture on *x*-ray which sometimes enables the physician to make the correct diagnosis. This picture resembles a bunch of grapes and is produced by a bunch of large-sized polyps, but no such finding was present in our case. The only other recourse was exploratory operation, a measure which would not only clear up the diagnosis, but would enable the surgeon to resect the tumor should the pathology warrant such a step.

The patient was accordingly prepared for operation and a laparotomy performed. Inspection of the anterior and posterior surfaces of the stomach revealed no tumor, but there were two enlarged glands at the pylorus near the lesser curvature. The stomach was then opened, but inspection of the inner surfaces revealed no tumor which looked like carcinoma. The wall of the stomach was very thick, the mucosa quite thickened and congested, and the inner layer was thrown into large folds so that it resembled prominent cerebral convolutions. This

condition was especially marked in the region of the pylorus and was most marked in the region of the greater curvature. The major portion of the distal part of the stomach was resected up to the duodenum, and a gastro-enterostomy was performed, thus removing the abnormal portion and providing an artificial opening for the food.

Histologic examination of the resected portion showed that the thickening involved chiefly the lower portion of the glandular layer and was due to a hyperplasia of the culdesac of the glands in the mucosa. This hyperplasia formed a dense, plaque-like part in which the acini of the glands were so serrated that there seemed to be a marked reduction in the interacinous tissue. The muscularis mucosa was everywhere intact and the acini were normal and did not involve the other layers. The surrounding mucosa was normal. An anatomic diagnosis of plaque-like adenoma of the stomach was made and a good prognosis given.

The subsequent course is of interest. The patient was seen five months later, and stated that all symptoms disappeared and that she had gained much in weight. *x*-Ray showed a gastro-enterostomy that functioned well and there was no evidence of retention. The patient was again heard from three years later and continued in perfect health.

Summary.—1. All cases of chronic diarrhea should have the stomach contents examined, as an achylia may be the cause of chronic diarrhea.

2. Benign tumor of the stomach should be considered in a case which has all the earmarks of carcinoma, but which has lasted for a number of years. Chronic inflammatory conditions of the stomach, such as tuberculosis or syphilis, should first be excluded.

3. The constant absence of occult blood from the stools after numerous examinations should lead us to bear in mind the possibility of a benign tumor of the stomach.

4. Exploratory operation should be recommended in doubtful cases, as such a step may lead to the discovery of a benign condition which may be removed with a resulting permanent cure as occurred in our case.

CLINIC OF DR. ROBERT SONNENSCHEIN

POST-GRADUATE HOSPITAL

A SERIES OF UNUSUAL EAR, NOSE, AND THROAT CASES

GENTLEMEN: We will today show you some interesting and perhaps atypical cases illustrating certain diagnostic and prognostic points. In the unfortunate absence of the patients who have passed away we will present the history and review the symptoms which the individual showed when he was presented to you some time ago. It is only by experience that we learn, and often it is from the cases in which we have unfortunate results that we learn the most. Anyone and everyone makes mistakes, but it is said that only a fool makes the same mistake twice. There is a saying, "Count that day lost whose low descending sun sees on thy part no worthy action done," and I would perhaps paraphrase it, "Count that work or that case lost from which you do not learn some important point which will guide you in your future work." Therefore, not only carefully examine every patient who comes before you, whether his ailment seems insignificant or grave, but weigh all the evidence presented by the symptoms, so that you will not only be aided in your diagnosis of future cases of a similar nature, but that if an unfortunate ending occurs you may be able at the postmortem to correlate your findings during life with the pathologic conditions noted at the necropsy.

Case I.—Miss A. H., aged thirty-seven, correspondent, came to us some months ago with a history of having had an acute suppurative otitis media on the right side for six weeks. There had been considerable tenderness over the mastoid process and a thick purulent discharge which was much more profuse

at first than when she presented herself to us. The patient had complained of very little pain and denied having at any time had any fever. Some time previously she had had some nasal operation performed to relieve obstruction to breathing. The patient stated that she had seldom experienced any infection of the throat.

Examination at that time showed a deviation of the nasal septum to the left. The tonsils were flat, but contained no pus, and the mucosa of the pharynx was quite red. In the examination of the ears the left one was found negative. The external auditory meatus of the right ear showed considerable swelling or, as it is often termed, "sagging" of the posterosuperior wall of the bony portion of the canal. The right mastoid process was tender and considerably swollen. Examination of the hearing for the unaccentuated whisper was 4 meters on the left ear, but only 10 cm. on the right or affected ear. The *x*-ray picture of the mastoid showed gross changes in the right one, with a breaking down of some of the cell partitions. There was no question at all in our minds of the presence of an acute mastoiditis caused by the acute suppurative otitis media. The patient, you will perhaps recall, was very apprehensive of any operative procedure. We considered seriously the significance of the two very marked changes present, namely, swelling of the bony portion of the external auditory meatus and the changes in and about the mastoid process. The indications for the acute mastoid operation vary with every otologist. Some men advise operating if there has been a purulent discharge from the ear which resists the ordinary measures for a period from four to six weeks, even though no other symptoms, such as fever, marked pain in or about the ear, tenderness or swelling of the mastoid process, or changes in the external auditory canal are present. They proceed on the basis that a long-standing suppuration, even though it may not cause life-endangering complications, will irretrievably impair the hearing in the affected ear, and, therefore to preserve that function rather than to avoid some intracranial or other complication they advise opening and cleaning out of the mastoid cells. Other otologists, on the

other hand, feel that irrespective of the amount or duration of the suppuration, other symptoms and signs should be considered significant in the indications for mastoidectomy.

During the first week or so of most cases of acute otitis media there is very often fever (especially in children) of moderate degree, some tenderness over the mastoid process, especially in the region of the antrum or at the very tip of the mastoid in the highly pneumatized mastoid. The fever, pain, and tenderness in the uncomplicated cases, however, usually disappear at the end of seven to ten days. If so, and if then later on at any time the fever, pain, or tenderness reappear, or if we find swelling over the mastoid process with thickening of the periosteum, operation is usually performed by most otologists. If there is a swelling or sagging of the above-mentioned posterosuperior portion of the bony external auditory meatus, it is assumed that there is a periostitis in the region of the antrum, and this sagging is usually considered a definite indication for opening the mastoid process.

It is usually assumed that it takes from two and a half to three weeks in the suppurative process before sufficient decalcification occurs, so that the trabeculæ between the individual mastoid cells break down and a number of these cells coalesce to form various sized cavities. During the first week or ten days there is always in pneumatized mastoids an involvement of the mucosa lining the cells, so that an *x*-ray picture almost invariably shows a clouding of the cells as compared to those in the normal mastoid. This, however, does not mean very much, because it was shown by Politzer years ago that in almost all pneumatized mastoids there is an extension of the inflammatory process from the middle ear to the mastoid cells within the first day or two of the onset of the disease, but this does not mean that a definite mastoiditis will develop. In fact, most of these cases subside promptly. Of course, in those cases where there is either poor pneumatization or a practical absence thereof, there will not be this extension to the mastoid process, since there will be very few cells present. Where the pneumatization is greatly retarded the partitions between the cells are

usually very thick, and they will, therefore, not be likely to break down and give the picture noted radiologically in the well-pneumatized mastoid.

Bearing in mind this point and also the fact of the swelling and tenderness of the mastoid process plus the changes shown in the radiologic examination in this case, we shall see that our patient reacted differently from what we would have been led to believe.

You will recall that we explained these anatomic, pathologic conditions to the patient, and told her that usually one or both of them are definite indications for operative intervention. Owing to her reluctance, however, to have anything done aside from conservative local applications we were compelled to adopt an expectant attitude. Light inflation of the ear with the Politzer bag and the application of 50 per cent. alcohol dressings, together with requiring the patient to abstain from work and rest in bed, were the measures we outlined when she was before you at the time.

Very much to our surprise within two weeks' time there was a complete retrogression of the symptoms. The swelling in the external auditory meatus diminished and finally disappeared, the tenderness and swelling of the mastoid also subsided. Within ten days the hearing for the unaccentuated whisper in the right ear had increased to $1\frac{1}{2}$ meters as compared with 10 cm. previously noted. The suppuration had entirely ceased and the drum membrane showed only slight injection. At present, five months after the original acute infection, the patient's ear is to all intents and purposes normal, and the hearing of the whispered voice has increased to 4 meters, the same as on the well ear.

Here we had, despite the gross changes present, a complete subsidence, with apparently a *restitutio ad integrum*. Just what the condition of the mastoid cells on the affected side now is I do not know, since the patient declined to have any further radiologic examination made. There are at least two important lessons to be drawn from this and from our next case. The first is, that gross macroscopic changes, usually considered ab-

solutely indicative of the necessity for surgical intervention, may in some cases entirely subside with purely local palliative measurements. This should, however, not be erroneously evaluated, and one should not in the ordinary case presenting such symptoms assume that it will proceed favorably and that no serious complications will ensue if we simply watch and wait. It is necessary in all instances to use one's judgment based upon previous experience; and then be guided by the appearance of the patient, the presence of fever, the leukocyte count, and the fact as to whether the symptoms are getting worse. If these signs are unfavorable, operate, unless there is soon a very radical change for the better in the individual. The second point to be remembered is that we must not assume, as is so often done, "post hoc propter hoc," that the result we get is due entirely or largely to some specific measure applied. If, for example, in this case some enthusiast had used some new germidical preparation or some new mechanical contrivance, he would have assumed very probably that the recovery was due to that particular line of treatment. As a matter of fact, we simply used a light inflation of the ear with 50 per cent. alcohol dressings, which, as you know, we are in the habit of applying in many cases of acute otitis media with mastoid symptoms. Surely, it was the resistance of the individual's tissues that brought about the favorable result in this case and not any special line of medication. Be careful, therefore, not to be deceived by the fact that in any particular case or in a few cases a favorable result is obtained by the use of some new therapeutic measure, for it is just as likely that the particular case in point might have recovered with any other or no specific medication. The only way, it seems to me, in which we can assume that any particular line of medication gives a certain definite percentage of recoveries is by using it in a large series of cases with an equally large number of controls in whom the particular agency we are investigating is not employed, and then very carefully analyzing the results before endeavoring to arrive at a definite conclusion regarding the possible value of such medication.

Case II.—Mr. L. O. H., aged forty-four, automobile mechanic, came to us a month ago with a history that following a head cold six days previously he began the next day thereafter to have a very severe pain in the right ear. Thus far there had been no discharge from the ear.

Examination of the nose was negative. Tonsils were submerged, but contained no pus. Examination of the ears showed the left one negative; the right drum membrane was greatly injected and had a vesicle on its posterior half. The Weber test showed lateralization of the sound to the right or affected ear. Examination with the whispered voice gave hearing 2 meters on the right ear and 4 meters on the left ear. In view of the fact that the hearing was still quite good and that there was a vesicle present upon the drum membrane, we made the diagnosis of a myringitis bullosa. Ordinarily when there is any fluid in the middle ear we find the hearing of the whispered voice, especially for the lower tones, reduced to 1 meter or even less. When there is only a myringitis present the hearing sometimes is not very greatly reduced. The puzzling feature here, however, was the fact that the Weber was lateralized in the right or affected ear, which is usually true when there is any involvement of the conduction apparatus, and especially when there is any fluid in the middle ear, either of a serous or purulent character. In most cases of a simple myringitis or involvement of the drum membrane itself we have found that the tuning-fork is not definitely lateralized in the affected ear. On the first day we saw the patient we merely incised the vesicle without cutting through the drum membrane itself. Eight per cent. phenol glycerin pack was inserted into the right ear and sodium salicylate in 5-grain doses given every four hours.

When the patient returned to the clinic the next day the condition had become worse and the hearing had diminished. Under gas anesthesia a wide paracentesis of the drum membrane was then carried out. Very shortly thereafter the ear began to drain, at first a serosanguineous fluid, which within twenty-four hours became purulent. Despite the free drainage the patient continued to have for two weeks very severe head-

aches, especially in the right parietal region, which almost entirely prevented sleep. An *x*-ray picture showed a definite infiltration of all the cells, but, of course, at this time no breaking down of the trabeculæ. The patient apparently had a very hard mastoid cortex. The leukocyte count at this time showed only 6600 cells.

Severe headaches are usually not present in the ordinary acute otitis media patient, and especially not when there is free drainage. When these headaches are severe and cause insomnia, as they did in our patient, they are usually of grave import. Very often they indicate an irritation of the dura due either to an erosion of the bone and exposure of the dura, or to a subdural abscess. When these headaches are persistent, are increasing in severity, and defy the ordinary narcotics and hypnotics, it is usually necessary to operate promptly in order to prevent, if possible, serious intracranial complications, and if a subdural abscess is already present, to evacuate the pus.

Owing to the history of the severe headaches, the *x*-ray findings, and the appearance of the patient (who by this time through the pain and loss of sleep together with the consequential loss of appetite began to look very bad) we advised admission to the hospital for careful observation, and if then within a day or two no definite and speedy improvement, opening of the mastoid process. The patient, as you will recall, was a most obstinate individual, who insisted that he certainly would get well by the ordinary treatment, and even wished to go on with his work despite the loss of sleep, etc. Not being able to get his consent to enter the hospital, we had to content ourselves with treating him symptomatically by giving pyramidon for the pain and luminal for the insomnia, together with the application of 50 per cent. alcohol dressings. He was right and we were wrong, for within three and a half weeks the pain had disappeared, the ear was dry, and the patient's appearance had materially improved. You see him today a well man.

In this case, like the one just before, we have seen a recovery without operative procedure where we had every reason to assume that some intervention would be necessary. It is diffi-

cult to explain the intense and very persistent headaches which cause insomnia, anorexia, etc., on the basis of an extradural abscess in this case, for that certainly would not have been likely to recover spontaneously. Some dural irritation was present, but how it was incited we cannot tell in this patient because no biopsy was performed. It is true, however, that in most cases of severe headaches in acute otitis media one must consider very seriously the possibility of intracranial complications, and if the headaches do not subside promptly and the other symptoms of the acute otitis media persist, it is usually necessary to operate. Let us emphasize once more the fact brought out in the discussion of the previous case, that we must not draw hasty conclusions regarding the efficacy of one or the other particular line of treatment simply from the fact that one patient with a certain set of symptoms recovers. We used as outlined only the very simplest symptomatic measures which certainly could not be considered specific.

Case III.—Mrs. R. C., aged forty-five, widow, came to us a year ago with a history of a so-called "pounding tinnitus" chiefly in her right ear, but without impairment of hearing. At no time had there been any suppuration. The patient occasionally had "head colds" and rather frequent attacks of tonsillitis, especially in the wintertime. On this occasion you will perhaps recall we merely catheterized the ears and gave her potassium iodid and bromids in the endeavor to relieve the tinnitus aurium.

Examination of the ears showed some soft cerumen, which was removed, and some dulness of the drum membranes, more so of the right one. The hearing for the unaccentuated whisper was 5 meters in both ears. At that time we advised the patient to have a tonsillectomy in case there was frequent recurrence of the tonsillitis. She returned to us last week after a lapse of over nine months. The report from the internist was that her blood-pressure was rather low, only 98 systolic, that her teeth were rather bad, and that her urine showed considerable albumin. He advised tonsillectomy.

Examination of the tonsils at this time showed them partly submerged, with a great deal of caseous purulent material in the crypts. The patient was sent to the hospital in the afternoon and the operation was planned for the next morning. We saw her at about 8 o'clock in the evening, at which time she seemed to be feeling perfectly well. When the nurse went to her at 5 o'clock the following morning to take her pulse and temperature she found her dead in bed. Unfortunately, only a partial postmortem was obtainable, namely, of the neck and chest, but the pathologist found nothing which would account for the sudden and unexpected death. Whether cerebral embolism or some other intracranial condition was the cause of this fatality we do not know. Fortunately for us the death occurred a few hours before the operation which had been planned. Had the death ensued at any time after the operation or even within a week or two thereof, the laity would have accused us of being the cause of the fatality. We realize, of course, that many patients or at least a goodly number have died during operation, either during a general anesthetic or under a local one. The use of cocain and adrenalin is fraught with many dangers, and we in this clinic have entirely discontinued for a long time the use of cocain even for light topical application in cases of tonsillectomy. By means of nerve blocking with novocain (procain), aposthesin, or other substances excellent anesthesia is obtained with very little if any danger. Adrenalin in very dilute form is used sparingly in these nerve-blocking injections. The addition of alcohol to the anesthetic fluid (giving them a concentration of 25 to 33 per cent.), injected into the glossopharyngeal plexus, has in our hands not only prevented syncope in practically all cases, but less postoperative pain.

As in our first cases, so here we can also say that one should not judge too definitely the results of one line of medication, for the death occurred before any operative or medical intervention. Had it happened a few minutes or a few hours after the use of some local anesthetic all blame for the fatal issue would have been placed on the particular anesthetic or method used.

Case IV.—Miss S. S., aged fifteen, student. This young lady, you will recall, came to us recently complaining of pain above both eyes, together with considerable greenish-yellow secretion, especially from the right side of the nose. There was considerable tenderness on palpation in both supra-orbital regions. The patient stated that she had been subject to frequent "head colds" and also rather frequent infections of the throat, the last attack of which was a month ago and was quite severe. She claimed that the condition then present had existed for five or six days. On examination of her nose we found at that time deviation of the septum to the right, with pus in both middle meati, together with a polypoidal left middle turbinate and a polyp in the left middle meatus. In the nasopharynx there was also much pus which had dropped down from the nose. The tonsils were flat and submerged and contained no pus. Transillumination of the sinuses showed that the frontals and maxillary antra were quite dark.

The treatment applied was cocainization of the middle meati and irrigation of the antra with a very dilute copper sulphate solution by means of a cannula passed into the natural openings in the middle meati. In this way an enormous quantity of pus was evacuated, especially from the right antrum. To empty the frontal sinus nasal suction was employed. To relieve pain the patient was instructed to use pyramidon in 5-grain doses every four hours if needed, to introduce into the nose by means of an atomizer a solution containing $\frac{1}{2}$ per cent. antipyrin, and 1 : 8000 solution of adrenalin chlorid. At this point let me say that we have been in the habit of using only the dilute solution of adrenalin chlorid, especially in acute nasal conditions, preferably the 1 : 8000 concentration, or in infants, 1 : 16,000. If you use a 1 : 1000 or 1 : 2000 solution you get a very marked constriction of the tissues, with considerable relief for a very short time. Then, however, when the effect of the adrenalin has disappeared, there is a very marked reaction, with considerable swelling of the mucosa, and often the patient is more uncomfortable than he was before the use of the astringent. When the more dilute solutions, as indicated

above, are employed a much more lasting benefit is obtained, due to the fact that there is not so marked a reaction following the use of the drug. The patient was also instructed to apply heat to the forehead and cheek after using the adrenalin spray. It is necessary always to caution patients against using too high a degree of heat, since burns of the skin often occur where hot-water bag or electric pad is used without due caution, and the resulting burns are more painful and discomforting to the patient than is the original condition for which the heat is applied.

In order to get better drainage from the antra we instruct the patients to lie on the side opposite to the one involved with the head inclined a little downward, so as to bring the ostium of that antrum as low as possible and facilitate drainage. For the frontal sinuses the erect position is good because the drainage is then directly or almost directly downward. In the case of the maxillary sinus the point of exit with the head erect is far above the floor of the antrum, hence the drainage must be against gravity, which is, of course, very difficult, and will only occur when the cavity is practically filled to overflowing.

For two days the patient seemed to improve greatly, the pain lessened to a marked degree, there was free outflow of pus, and no fever was present. Suddenly, however, things took a change for the worse. We were notified early in the morning a few days ago that the patient had had a very severe headache during the night and that there was some rigidity of her neck. We ordered her at once transported to the clinic, where she was examined and the following symptoms noted: The pulse was rapid, the temperature was 102° F. per rectum, there was considerable rigidity of the neck, and a decided Kernig reaction. Spinal puncture showed fluid cloudy under high pressure with about 6000 cells to the cubic millimeter. There was considerable tenderness over both frontal sinuses and the patient was irrational. In a word, we had the picture of a diffuse cerebrospinal meningitis apparently due to an acute sinusitis. Consultation was held, and it was decided that nothing could be gained by any operative procedure in so fulminating a case of

diffuse meningitis. Within less than twenty-four hours the patient was dead. Despite all efforts permission for postmortem examination could not be obtained from the parents.

We know, of course, that by means of the lymphatics running through the cribiform plate of the ethmoid communication between the nasal cavity and that of the cranium exists, and it is in this region that infections so often travel from the nose to the meninges. In this case, where we had apparently free drainage both from the frontals and the maxillary sinuses, one would not have expected this serious complication to occur. However, the patient no doubt had an involvement of the anterior ethmoidal cells, and it was probably from this region that the infection spread to the cranial cavity. The nature of the infective agent must have been exceedingly virulent since death occurred so soon (twenty-four hours) from the beginning of the first alarming symptoms, namely, the intense headache and the beginning stiffness of the neck. Therefore, never lose sight of the possibility of very serious, even fatal, complications arising in the course of acute infections of the nasal accessory sinuses. Where there is obstruction to drainage with a backing up of the purulent secretion the probability of extension to the cranial cavity is much greater than where there is apparently ample drainage, such as we thought we had in our patient.

Case V.—B. S., aged sixteen, student, is another example of serious complication resulting from acute sinusitis. When we first saw this patient there was the history that two weeks previously one of the upper left teeth which had been filled a year before caused a swelling in the palate. The swelling was later opened by a dentist one week ago. About this time the patient had been swimming and contracted what he thought was an ordinary "head cold." Two days before his appearance at the clinic the sight in the left eye seemed blurred and a very severe left frontal headache appeared. A purulent nasal discharge began at this time.

Examination showed large middle turbinates, especially the left one, which was quite boggy. There was much pus in the

left middle meatus, with considerable edema of the left eyelid and of the conjunctiva.

Under cocain anesthesia the left middle turbinate was infracted away from the lateral nasal wall, followed by a tremendous outpouring of pus. Nasal suction, application of heat, and the use of the adrenalin spray were ordered. Examination of the mouth showed a swelling in the left half of the hard palate, and from the incision which had been made by the dentist some dark bloody fluid was escaping.

For two days the patient seemed to improve, but then there was an increase in the swelling of the eyelid, the headache became more intense, and there was some rise in temperature. Under ether anesthesia the left middle turbinate was snared off and the ethmoid cells easily opened with a curet. Then an external incision was made through the left eyebrow and via the anterior wall the left frontal sinus was opened and a small amount of very foul-smelling pus evacuated. With a curet the nasofrontal duct was enlarged and the ethmoids further opened. For several days the patient seemed to improve, but it was necessary to incise the left eyelid to permit the escape of pus, which was very foul smelling. At this time the patient had a slight convulsion and there was a sudden rise in temperature. The wound over the frontal sinus was reopened and the posterior wall of the sinus removed; the dura looked rather gray, but was not bulging. We felt it would be advisable to incise the dura and explore the frontal lobe, but the neurologist present at the operation advised against further procedure at this time. Two days later, however, the patient became irrational and very restless, the packing was removed from the wound, the dura incised, and the left frontal lobe entered. There was an escape of a very large amount of pus and the patient became conscious very shortly thereafter. However, within a few hours he again lapsed into coma and died two days later. In this case also we were unable to obtain postmortem examination.

Here we have another instance of fatal intracranial complications following closely upon an acute sinusitis. In the former case there occurred a diffuse purulent cerebrospinal meningitis.

In the second case we have a large abscess in the frontal lobe following an acute involvement of the frontal sinus and the anterior ethmoidal cells. In the first case there was a history merely of a head cold. In the latter case there was the distinct history that the nasal infection followed closely upon the swimming. As a matter of fact, we know that many of the infections of the sinuses following the entrance of water into the nose while swimming or bathing are of a very virulent nature and are frequently accompanied by serious complications of one kind or another. It has been our experience, furthermore, that in the frontal lobe abscesses following acute sinusitis the prognosis is very bad even though the abscess is found and evacuated comparatively early. Some of the cases improve for a few days or a week or even longer, and then develop an encephalitis which causes death even though there had been good and ample drainage of the original abscess. When there is, on the other hand, a brain abscess of a chronic nature there is time for the formation of a dense capsule which often prevents the extension of the process and the causation of the fatal encephalitis. Our regret in the second of these cases was the fact that we did not incise the dura on the day when we had exposed it and explored the frontal lobe. I am sure the abscess was already quite large at that time, and there is the possibility, but only the possibility, that we might have saved the patient. As it has been said before, "of all sad words of tongue or pen the saddest are these, it might have been." Much as we regretted not having opened the abscess earlier, we realize that the probability is that even though we had done so the end-results would very likely have been the same. The first principle of surgery, however, is that when there is a collection of pus anywhere, to open it and drain it as quickly and as thoroughly as possible.

Case VI.—N. G., a boy aged nine and a half years, student, had a severe influenza two months ago from which he apparently made a complete recovery. One symptom, however, remained, and that was a severe pain of a rather intermittent character which occurred every few hours and lasted for from four to ten

minutes. When the pain was absent, examination of the ears showed them entirely negative. The hearing for the whispered voice was 7 meters. There was no tenderness of the tragus, the mastoid region, or of the infra-auricular glands. There was no redness or swelling of the external auditory meatus or of the drum membrane, but during the attacks of pain his mother tells us that he was exquisitely tender, especially in the region of the tragus, and would not tolerate the slightest contact of any sort either of the hot-water bag, electric pad, or even a layer of cotton intended to keep the ear warm.

Examination of the nose was entirely negative, as was also the pharynx. The tonsils had been removed some years ago. We learned that this patient had had in previous years several attacks of acute otitis media requiring paracentesis each time. The drum membranes, however, had healed perfectly and the hearing, as previously noted, was perfect. Where an external otitis is present we usually find tenderness at one or several points; either at the tragus, at the insertion of the auricle (and this is usually noted when the auricle is moved or pulled upon), and lastly, tenderness just below the external canal in the location of the infra-auricular gland. This was entirely absent in our case. Where an acute otitis media is present we usually have some or considerable injection of the drum membrane, with a loss of the landmarks and often with bulging. At the same time there is a diminution in hearing and there is often some tenderness over the mastoid process, especially in the region of the antrum. All these signs and symptoms were absent in this little boy. He is very intelligent and described the symptoms in detail and very accurately. One had to consider the possibility of referred pain from the teeth, for we know that pains in and about the ear are often due to involvement of some structure not resident within the ear. This otalgia may be due either to a dental caries, to inflammatory processes in the pharynx, especially in the region of the tonsils (either a tonsillitis or a peritonsillar abscess), or it may be referred from a laryngeal ulceration, especially where the lesion is located on the posterior wall of the larynx. The fifth cranial nerve directly or via com-

munications supplies this area. Bearing these facts in mind, a thorough examination of the pharynx, larynx, sinuses, and teeth was made. The latter were *x*-rayed, but absolutely no pathologic condition anywhere was found. The pediatrician in consultation could find no cause for the trouble and simply advised "watchful waiting." It occurred to us that there was the possibility of a neuritis or involvement of Meckel's ganglion of a toxic nature due to the influenza, which might be responsible for the sharp pain. On that basis the ganglia were cocainized and then a very dilute formalin solution applied. At the same time to act as an alterative potassium iodid in 5-grain doses was ordered three times a day. Much to our gratification a few applications to Meckel's ganglia gave the patient complete relief, and he is today as you seen him entirely free from all symptoms.

One should not forget the fact that many infectious diseases, such as influenza, typhoid, scarlet fever, and others, often cause a neuritis in one part of the body or another, and that many of these cases stubbornly resist treatment. Whenever a pain is present it is merely a symptom and not a disease. You recall that we have emphasized this fact many times with reference to headache. Do not merely treat the pain with sedatives (this, of course, may be necessary as a palliative for the time being), but endeavor, if possible, to find the etiologic factor. Often this will be an ulceration in the pharynx or larynx, an abscessed or carious tooth, a sinusitis, a brain tumor, or, it may be, as it was in this case, apparently a neuritis following an acute infectious disease.

Case VII.—Miss E. B., aged fifteen, student, came to us six weeks ago with the history that since infancy, following measles, she had had an intermittent discharge from the left ear. One week before presenting herself she had a severe sore throat, with fever and slight pain in the affected ear. The day before we first saw her there suddenly appeared a paralysis of the entire left side of the face, first observed by the patient, in that she could not move her left facial muscles at all. On examina-

tion we found that all branches of the left facial nerve were affected, and we concluded that a peripheral lesion was present because a central one would probably not have involved all branches at once. The nose showed a septal crest low on the left side. The tonsils had been removed five years before, but the pharynx showed considerable lymphoid tissue which was thickened and hyperemic. In the neck there were some palpable lymph-glands. The right ear was entirely negative. The left ear showed a loss of about two-thirds of the drum membrane. Only the upper third remained. There was a slight amount of secretion of a foul character in the tympanic cavity and a considerable amount of granulation tissue.

The case was referred to the neurologist, who confirmed the diagnosis of a peripheral lesion, but found that the muscles reacted very well to the opening and closing of the galvanic current. The question which we had to consider seriously at the time was whether it was proper or necessary to at once operate because of the facial paralysis. You will recall that the facial nerve runs along the inner wall of the tympanic cavity between the horizontal semicircular canal and the oval window, then entering the fallopian canal, it passes downward and outward, finally leaving the temporal bone at the stylomastoid foramen. Now it occasionally happens that there is a congenital dehiscence or defect in this bony fallopian canal, so that when an acute otitis media occurs, the nerve is directly pressed upon by the secretion, and at once a complete facial paralysis or at least a paresis appears. In these cases it has usually been considered the best surgery at once to operate, open the mastoid process and the antrum in order to facilitate quick and ample drainage, and thus endeavor to relieve pressure upon the nerve. This we have done in a number of cases with very excellent results, in that there was complete restoration of function of the facial nerve within five or six weeks. In some cases instead of a dehiscence being present there is an erosion of the bone by the pus, with consequent involvement of the facial nerve. Here, of course, the same principles of surgery apply. In our case, however, we had the distinct history of a very chronic condition

present throughout the life of the patient from earliest infancy to the present age of fifteen. Had there been a congenital dehiscence of the fallopian canal we should have expected that when the condition of acute infection of the middle ear was originally present in the course of the measles which caused it, the symptoms relative to the facial nerve would then have promptly appeared. Instead of that we had the suppuration for almost fifteen years before the onset of the paralysis. On the other hand, it was possible that an erosion of the bone might have been caused by the chronic suppurative process. In view of the fact, however, that in this case there was a very large perforation of the drum membrane, so that a retention of pus in the middle ear was not at all likely, and, therefore, marked pressure on the fallopian tube, with consequent erosion very remote, we felt justified in assuming that some other factor was the cause of the facial neuritis, if such it was. You will recall that we told you in the early part of the recital of this case that the patient had been suffering for a week before the onset of the facial palsy with a severe sore throat accompanied by fever. In view of these symptoms we believed that the facial nerve involvement was of a toxic character due to the throat infection. On that basis we applied guaiacol in 25 per cent. solution to the pharynx and the neurologist treated her with faradism. At the same time we merely kept her ear clean and within a few days the suppuration had almost disappeared. Soon after the electric treatments were instituted some improvement was noted, and you now see the patient six weeks later with practically perfect restoration of function in her facial muscles.

Here was a case in which a most momentous decision had to be made—whether to operate or not. When to operate, and during operations knowing when to stop, are two principles much more difficul to acquire than mere operative technic. The first inclination here, as also in the acute cases, would be to operate, but we must remember that the mere presence of pus in the cavity did not mean that it alone was the cause of the condition present. That this was of toxic origin and not due to

pressure or erosion is proved, I believe, by the further course of the case.

We have been able to present only the records of some of our cases, since they unfortunately have passed away. Having seen them as you did during life, a résumé of the histories and the course of these sad cases should be instructive. It is from the failures that we often learn more than from the successful cases. In the latter we often assume that what we have empirically or rationally done for the patient is the measure which has caused his recovery. We often lose sight of the fact that in medicine with very few exceptions our efforts are largely effective in that they assist patients to recovery. It is the natural resistance of the individual that is a very important factor in all restorations to health. In a few classes of cases operative procedures are, of course, mainly if not wholly responsible for the recovery of the patient, in that we either provide drainage for suppurative processes when we remove directly infected or necrotic tissue, or in that we relieve obstructions to breathing and other functions. In those instances, however, where we have death of the individual despite all medical or surgical procedures at present known to medical science, we are impressed with the fact that there is a colossal amount of work to be done in the endeavor to find, first, the cause, and second, the proper remedial agent to prolong the patient's life. Furthermore, these sad cases teach us not to be too reliant upon the measures now at our disposal, nor to make too favorable a prognosis in cases that have certain serious aspects. We must carefully study all our cases from every angle and then apply the most rational measures at our command.

CLINIC OF DR. LAWRENCE E. HINES

NORTHWESTERN UNIVERSITY MEDICAL SCHOOL
AND ST. JOSEPH'S HOSPITAL

CARDIOVASCULAR SYPHILIS

THE Treponema pallidum produces such a variety of lesions in the heart and aorta, that any attempt to generalize the clinical picture produced by these lesions is difficult and sometimes misleading. Correlation of symptoms with necropsy findings in cases peculiar only to themselves is usually a more instructive method of study. One case is chosen for detailed presentation because it seems to allow an accurate interpretation of a group of symptoms of cardiovascular syphilis.

Case Report.—A married man, thirty-two years old, was first observed February 2, 1924. His chief complaint was substernal pain, described as a continuous dull ache, in an area the size of his hand, beneath the upper part of the sternum. Other complaints were slight cough, slight hoarseness, dyspnea on exertion, and weakness.

Twelve years ago he had a chancre of the penis, but no skin eruption. He received practically no treatment for the chancre. He had no other illnesses. He married six years subsequently; his wife was well; there were no children or miscarriages.

The present illness started abruptly nine months ago, with a sudden attack of severe, agonizing substernal pain which was associated with dyspnea, sweating, and terror. The first attack lasted about fifteen minutes, and since he has never been free entirely from annoying substernal sensations, either in the form of severe pain, a dull ache, or an oppressed tight feeling. The physician who first treated him found a positive Wassermann test of the blood-serum, and gave him a course of 6 neo-arsphena-

min injections, and subsequently mercury inunctions and iodids. Slight improvement followed, but never since the onset did he feel able to continue his usual occupation, that of a chef. About five months after the onset the pain became more severe, agonizing attacks became more frequent, and the pain radiated to the right shoulder and arm. The administration of iodid combined with digitalis caused some improvement.

When I first examined him he was feeling about as well as he had at any time since he first became ill. He was taking 15 drops of tincture of digitalis three times daily and 10 drops of the saturated solution of potassium iodid three times daily. Physical examination revealed a large, well-developed man who was dyspneic on slight exertion. Pallor of the skin and mucous membranes was marked.

Eyes.—The scleræ were white; pupils equal, round, regular, and reacted to light and accommodation.

Nose and Ears.—No gross changes.

Buccal Cavity.—The teeth were clean and in good dental repair. Tonsils were small.

Neck.—Bilateral carotid pulsation was pronounced.

Lungs.—There were no abnormal findings except for a few moist râles at the base of both lungs, posteriorly.

Heart.—The apex-beat was in the sixth interspace, 12 cm. from the midsternal line; the upper border in the second interspace; left border in the axilla 13 cm. from the midsternal line; right border 3 cm. from the midsternal line. A loud systolic and a louder diastolic murmur was heard over the base of the heart, with greatest intensity in the third interspace to the left of the sternum and transmitted to the vessels of the neck and over the entire precordium. The superior mediastinal dulness in the second interspace was 9 cm. wide. The pulse was of the collapsing type, the so-called Corrigan pulse. The rate was 88. The systolic blood-pressure was 110, the diastolic 50.

Abdomen.—The liver could be palpated below the costal margin; the spleen could not be felt. There were no areas of tenderness.

Extremities.—There was no edema. A capillary pulse could

be seen in the nail-bed of the fingers. The inguinal, axillary, epitrochlear, and cervical lymph-nodes were palpable.

The urine showed only a small trace of albumin.

Progress.—2/4/24: Neo-arsphenamin 0.15 gm. intravenously.

2/10/24: Neo-arsphenamin 0.3 gm. intravenously.

2/17/24: Neo-arsphenamin 0.45 gm. intravenously.

2/25/24: Patient's symptoms were aggravated. For the first time he had developed edema of the ankles. The liver was increased in size and was tender. The right heart border was 4.5 cm. from the midsternal line. There was a large amount of albumin in the urine and a few granular casts. The neo-arsphenamin injection was not given. He was given pulv. digitalis, gr. 2, every four hours.

3/3/24: No improvement. He was unable to sleep because of the severity of the substernal pain. There was no radiation of the pain. He had slight attacks of hemoptysis, severe dyspnea, moderate cyanosis, and marked edema of the legs and scrotum. He refused to enter a hospital. Morphin sulphate, gr. $\frac{1}{4}$, was prescribed for sleep.

3/6/24: Entered St. Joseph's Hospital because pain was becoming unbearable. Pain continuous and severe, and several times daily there were paroxysms of pain of an anginal character, associated with extreme dyspnea, ashy countenance, marked prostration, excessive perspiration, and the fear of impending death.

Treatment and Course in Hospital.—Morphin sulphate was given freely and tincture of digitalis, 15 minims, four times daily. A diet with protein limited to 60 grams daily, fluids limited to 1500 c.c., and salt restricted. Free purgation was obtained by large doses of heavy magnesium oxid. These measures failed to restore compensation. Nitroglycerin caused no relief of the pain. Two days after admission the clinical evidences of right heart embarrassment (dyspnea, hemoptysis, and extension of right heart border) prompted a venesection, with removal of 500 c.c. of blood. This seemed to relieve temporarily the dyspnea. He died February 18, 1924, and for twenty-four hours preceding death the precordial pain was so intense

and persistent that six doses of morphin sulphate, gr. $\frac{1}{4}$, gave no relief.

Laboratory Findings.—Blood-pressure:

	Systolic.	Diastolic.
3/ 8/24:	150	50
3/12/24:	140	30
3/17/24:	135	30

Blood Wassermann positive (4 plus).

Blood Chemistry:

3/ 1/24: Non-protein nitrogen, 68 mg. per 100 c.c.
Creatinin, 3.5 mg. per 100 c.c.

3/12/24: Non-protein nitrogen, 85 mg. per 100 c.c.
Creatinin, 4.2 mg. per 100 c.c.

Hemoglobin, 75 per cent.

Erythrocytes, 4,710,000.

Leukocytes, 8100.

Neutrophils, 68 per cent.

Large mononuclears, 2 per cent.

Lymphocytes, 29 per cent.

Transitionals, 1 per cent.

Temperature: There was never any fever, the temperature ranged from 95.6° to 98° F.

Pulse varied from 80 to 110, and was always regular.

Respirations were seldom less than 28 and as high as 40 per minute.

Necropsy.—The anatomic diagnosis is: Chronic adhesive and acute hemorrhagic pericarditis; syphilitic mesaortitis and myocarditis; saccular aneurysm of the posterior wall of the ascending aorta; syphilitic endocarditis of the aortic cusps; recent and old myocardial infarcts; multiple thromboses of the small branches of the coronary arteries; marked hypertrophy of all heart chambers; marked dilatation of the left ventricle; multiple, healed, chancre-like scars of the penis; recent infarcts of the kidneys and spleen; edema and extensive parenchymatous degenerative changes of the kidneys; edema of the lungs; right hydrothorax; ascites; general anasarca; chronic passive congestion of spleen, liver, kidneys, and intestines; healed tuber-

culosis of right lung; right fibrous pleuritis; healed scar of left hip; old healed left inguinal scar; recently healed surgical scar of left cubital fossa; fetal lobation markings of the kidneys; tattoo marks of the forearms; cyanosis.

The detailed findings of the heart are: The heart is huge in size and weighs 740 grams. The apex is made up entirely of left ventricle. The pericardial sac is obliterated by scattered fibrous adhesions and thin, easily torn, hemorrhagic fibrinous adhesions. When the outer sac is reflected a shaggy purple surface mottled with yellowish-gray opaque patches is seen. The yellowish-gray patches are in some places surrounded by a narrow zone of hemorrhage. They extend into the heart muscle for varying depths. The small branches of the coronary arteries which traverse these areas are in many places plugged with thrombi. The circumference of the tricuspid orifice is 14 cm.; of the pulmonary orifice, 7.5 cm.; of the mitral ring, 12 cm., and of the aortic ring, 7.5 cm. The margin of the mitral leaflets is thickened and smoothly nodular. The cusps of the aortic valve are markedly thickened, rigid, retracted, and slightly nodular. Beginning about 3 cm. above the aortic ring is a saccular outpocketing of the posterior wall of the aorta about 5 cm. in diameter and 4 cm. deep. In the deepest and central portion of the sac the wall measures less than 1 mm. in thickness, so thin that light can be transmitted through it. The lining of this sac and of the arch of the aorta is greatly roughened and thrown up into irregular longitudinal folds. In many places there are yellowish, opaque, slightly elevated nodules, from 1 to 2 cm. in diameter. No calcification is present. A few small scattered fibrous scars are present on the surface of the left ventricle and in the interventricular septum.

Microscopic.—The most striking changes of the myocardium are seen in sections through the infarcted areas, which show almost complete necrosis of muscle and the presence of a granulation tissue with many newly formed capillaries and numerous polymorphonuclear cells. Organized and partially organized thrombi have occluded many of the coronary vessels. In one section canalization of an old thrombus has occurred (Fig. 119).

There are interstitial changes everywhere in the myocardium, the predominating change being perivascular collections of lymphocytes and diffuse lymphocytic infiltration between muscle-fibers. Occasional fibrous scars are present. In sections of the heart muscle and aorta stained by the Levaditi method

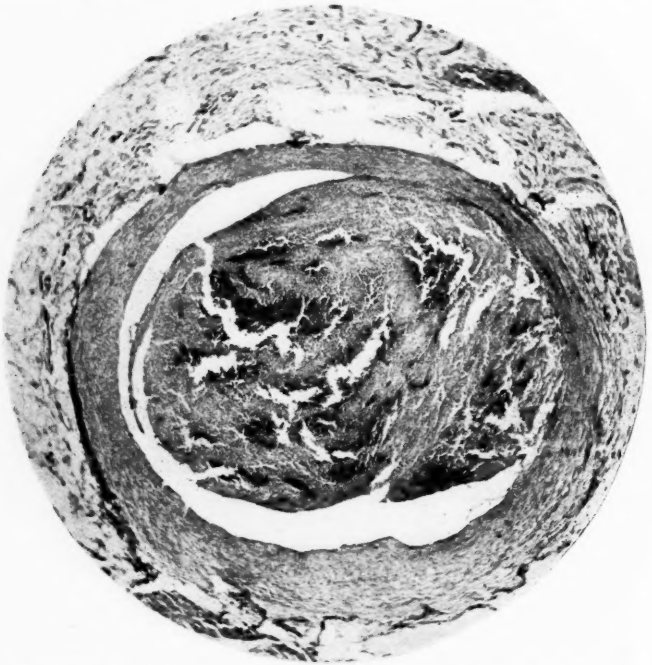


Fig. 119.—Thrombus in coronary artery with canalization of thrombus.

spirochetes are found in the inflammatory areas around the blood-vessels.

DISCUSSION

Etiology.—The cause of the extensive heart damage in this case is well established by finding Treponema pallida in the Levaditi stained sections of the myocardium. This confirms the clinical finding of a strongly positive Wassermann reaction.

Pathology.—This is an example of the usual and common

type of syphilitic lesions of the aorta. The damage to the ascending portion is greater, the transverse portion is involved to a lesser extent, and the descending portion is not diseased. In the posterior wall of the ascending aorta, where the greatest force from the column of blood is applied, is an aneurysm with marked thinning of the wall. Scattered over the surface of the diseased aorta may be seen yellowish, opaque, slightly elevated nodules which when sectioned are seen to be thickenings of the media. Another feature indicating the specific character of the lesions is the longitudinal folds and puckerings. The microscopic sections explain these findings as being due to patches of necrosis which have split the media in different places. The activity of the process is shown by collections of lymphocytes around the vasa vasorum, and especially by the presence of Treponema pallida in the wall.

The nodular and rigid aortic cusps are unquestionably incompetent.

Although the very striking myocardial changes which this heart presents are not rare, it is not common for syphilis to produce coronary thrombosis and infarction. These infarcts are very numerous, especially over the anterior surface of the left ventricle. Some are covered with a veil of hemorrhagic fibrinous adhesions. When the infarcts are incised we can see grossly that the small coronary vessels are plugged with thrombi. The fact that most of these thrombi are organized indicates that they are of considerable duration.

A recent communication by Wearn,[1] who analyzed 19 cases of cardiac thrombosis that came to autopsy and found none to be on a syphilitic basis, emphasizes that this manifestation of syphilis in the heart is unusual.

Scott,[2] however, who found coronary obstruction in one-fourth of his cases of heart failure due to cardiovascular syphilis, believes this is not so uncommon as formerly believed.

Symptoms.—Retrosternal pain was the most persistent and

[1] Wearn, Amer. Jour. Med. Sci., 165, p. 250, 1923.

[2] Scott, R. W., Jour. Amer. Med. Assoc., 82, p. 1807, 1924 (Abstr. of Meeting of Assoc. of Amer. Phys.).

distressing symptom which this man presented. He first sought medical advice because of pain; the pain prevented him from working in the last months of his life, and his death was preceded by twenty-four hours of terrific, agonizing pain, so intense that eight hypodermics of morphin brought no relief. Although aortitis and aneurysm, especially when they compress adjacent structures, frequently produce severe substernal pain, the intensity of the pain in this case, associated with collapse and fear of death, indicates that the pain, for the most part, was due to thrombosis of the coronary arteries. Although it is speculative to attempt to interpret the marked precordial pain at the onset, it seems likely that this was also due to thrombosis of the coronary arteries. This is suggested by the abrupt onset, the severity of the pain, the collapse, and the presence of old fibrous scars in the myocardium at autopsy. The organization of the thrombi is evidence that they were present a considerable time prior to death.

The visible pulsation of the carotids, high pulse pressure, the Corrigan or collapsing type of pulse, the physical signs of a huge heart, need not be emphasized as the typical findings of an incompetent aortic valve. And in like manner, the edema, cyanosis, enlarged spleen and liver, were evidences of a weak heart muscle.

Treatment.—In treating a case of cardiovascular syphilis our aim is to treat the syphilis and conserve a damaged heart. If we are unable to check the activity of the syphilitic process, our efforts to conserve the damaged heart, obviously, are of little avail. In the present case the usual methods of treating syphilis were employed, but the inadequacy of the treatment is clearly demonstrated by the progressively fatal course of the disease, the presence of Treponema pallida in the heart muscle, and the usual inflammatory changes produced by these organisms when active.

Of course, the ideal plan is to treat early syphilis so intensely that advanced lesions may not develop, but this ideal has not obtained, and the problem will still confront us. There is a great need, here, for more clinical investigation.

CLINIC OF DR. JULIUS H. HESS

COOK COUNTY HOSPITAL

DIAPHRAGMATIC HERNIA (EVENTRATION, TRUE AND FALSE HERNIA) IN INFANTS AND CHILDREN

ALTHOUGH diaphragmatic hernia is far from common, it was not until the Roentgen ray became generally used that it was possible, with any certainty, to make a clinical diagnosis in early life.

First in importance in the recognition of these cases is the diagnosis of an anomalous condition of the diaphragm which permits an upward displacement of one or more of the abdominal organs, with secondary compression and dislocation of the thoracic organs. However, retardation in development of the chest viscera during fetal life must be considered as a possible primary factor in some cases.

Secondarily, but of equal importance, with the diagnosis of pathology of the diaphragm, is the type and degree of deformity, upon which must be based an opinion as to the hope for life primarily, and incidentally the possibilities for surgical correction.

Diaphragmatic hernias are usually classified as congenital or acquired. The term "acquired" is undoubtedly erroneously applied in many of the cases, as it should be conceded that many of the cases presenting symptoms later in life result from earlier congenital weakness.

In these the diaphragm remains intact with a thinning of its structure; however, without the development of a hernial ring and sac In its stricter sense the term "hernia" should, therefore, be limited to those cases in which there is a protrusion of abdominal viscera through congenital or acquired openings. This takes place either through one of the preformed foramina or,

owing to deficiency or weakness of the muscle bundles, a weak spot exists through which the abdominal viscera are forced. The latter is facilitated by the pressure from below and the state of latent negative pressure existing in the thoracic cavity. The term "false hernia" is applied to those cases in which the abdominal organs have entered the thorax through an actual aperture in the diaphragm, either natural or traumatic in nature. In these cases there is an absence of a hernial sac and the structures are free in the chest cavity. In those cases described as "true hernia" the term indicates the presence of a sac in most instances composed of peritoneum and pleura.

Pathologic Anatomy.—Most of the reported cases have been left-sided, which in all probability is due to the large liver acting as a protective barrier on the right side. The diaphragm is a musculofascial structure, with a peritoneal surface below and a pleural surface above. It has several regions which have a tendency to develop abnormally because of their complex structure. Among these are the openings for the transmission of the esophagus, the aorta, the vena cava, the splanchnic nerves, and anteriorly the structures at the sternocostal junction. In most of the cases in which the hernia is present at birth it is at the seat of the foramen of Morgagni in front; that is, at the sternocostal junction or the quadrilateral space situated dorsally between the lumbar and costal regions. The hernial contents are usually the stomach and some of the intestines, although any or all of the mobile abdominal viscera may enter the chest cavity.

Eventration of the diaphragm is, in reality, not a hernia, but a diffuse relaxation of one or both halves of the diaphragm, which may, in their clinical aspects, cause symptoms simulating hernia. The condition is most often congenital, but it may be developmental. The latter group may result from a congenital predisposition either due to degenerative changes in the muscle-fibers, an insufficient muscle development, or anomalies of or injury to the phrenic nerve. In most cases the etiology remains obscure. Birth injury of the phrenic nerve is, however, to be considered as important in a number of cases. We may, there-

fore, have the condition following primary aplasia of the musculature, aplasia or atrophy of the phrenic nerve with secondary muscle degeneration, or, probably less frequently, both conditions developing from a common cause. The degree of eventration varies greatly and may reach the level of the second rib. Sauerbruch has carried out deliberately the division of the phrenic nerve for the purpose of producing paralysis and elevation of the diaphragm in connection with his extensive thoracoplasties. He found that the diaphragm under such circumstances becomes elevated to a remarkable height, often to the level of the third or fourth rib. Partial relaxations involving only a portion of the left side of the diaphragm have also been described as diaphragmatic diverticula.

Bayne-Jones in 1916 tabulated 45 cases of eventration of the diaphragm, 44 gathered from the literature and 1 reported by himself. Only 4 of these cases occurred in infants. Of the latter group, 3 were on the right side and 42 on the left. As the diagnosis in practically all of the earlier cases was made at autopsy and later by autopsy or *x*-ray, it may be surmised that only a limited number of the actual cases have been recorded in the literature. In most instances these were cases of extreme degree, and it can easily be conceived that many of the cases of minor deformity have escaped detection because of the absence of localizing symptoms.

True Congenital and Acquired Diaphragmatic Hernia.—The presence of a hernial sac is the typical finding in this group. In the *congenital hernias* a complete closure of the septum transversum must have existed in order that the hernial sac may have developed. Such a sac is usually composed of a layer of peritoneum on the under surface and pleura on the upper surface. In most instances it is probably due to a protrusion into the thorax of the recently closed septum before the development of muscle sufficient to resist the pressure from below. In many of the cases the sac contains the stomach and part of the intestines, and at times the spleen, pancreas, and even the liver. Displacement of the heart and failure on the part of the lung to develop may be primary or secondary. In *true acquired hernias* the

protrusion is either through one of the natural foramina which undergoes dilatation, or at the seat of a circumscribed weakened area which gives way after birth.

False congenital diaphragmatic hernia results from imperfect closure of the septum transversum, so that there is a free communication between the thoracic and abdominal cavities at birth. The opening may be of almost any size; more commonly, however, in the viable fetus, only a portion of half of the diaphragm is involved, most often in the space between the lumbar and costal regions.

Symptoms.—The symptoms of diaphragmatic hernia present the widest variation. Many of the congenital cases result in premature expulsion of the fetus or early death of the infant. On the other hand, large hernias have been present without symptoms directly referable to interference with the contents of the chest cavity, the true condition being revealed by roentgenologic examination or at autopsy. It is, therefore, probable that the condition is much more common than the number of published cases would lead one to suppose, many being overlooked because of the absence of subjective symptoms. More commonly early in infancy there is noted a general retardation in the development; a more or less marked asymmetry of the chest; abnormal physical findings, with a hyperresonance over the opposite side, typical chest findings on the affected side, which are dependent upon the lack of development of the lung; interference with respiration; and usually a displacement of the heart. This latter is usually in the form of a dextrocardia due to the fact that by far the greater number of cases are left sided.

As I have noted these cases clinically the local symptoms have belonged to one of two large groups—those referable to the respiratory and circulatory systems, which in my own experience have been the more common, and those involving the abdominal viscera, in which there was evidence of acute obstruction of the stomach or intestines. It is quite evident that the clinical picture varies greatly in a given case, depending upon whether the child is seen during a quiescent period or at a time of visceral crisis

Many of the infants present a clinical picture simulating that of congenital heart disease, such as retarded development, cyanosis, and clubbing of the fingers and toes, with a tendency toward recurrent attacks of respiratory infection. Intermittently the patients develop attacks of vomiting, constipation, of varying degrees with or without abdominal pain, meteorism, and other symptoms of ileus. This latter group of symptoms may develop without definite evidence of incarceration. Associated with the abdominal symptoms one almost invariably finds evidence of respiratory embarrassment and venous obstruction.

Physical Signs.—In the cases in which the hernia is large the signs are usually significant and often conclusive, while, on the other hand, if the sac is small and centrally located the diagnosis may be very difficult. Due to the fact that most of the cases involve the left side of the diaphragm, cardiac displacement, which is almost invariably present, is to the right, and in large hernias resulting in the passage of the stomach and considerable of the intestines into the pleural cavity, there is often a complete dextrocardia. The signs elicited by the presence of foreign organs in the chest cavity will vary greatly, being dependent upon the type of organs, their contents if hollow viscera, and the position of the patient. With an atrophied lung and chest filled by an empty stomach and intestines the chest may be tympanitic throughout. When the hollow organs are partially or wholly filled with a fluid content, on the other hand, the chest may be almost flat throughout, and when partially filled corresponding findings are to be expected. In no other condition do physical findings change so completely with the position of the patient, the administration of food, and the presence of complications.

When there is still some active lung tissue the expected resonance will be in direct relation to the site of the lung tissue. Breath sounds may be expected over the active lung tissue and at the edges of the area where the lung comes in contact with the hollow viscera. At this latter point the respiratory murmur may have a metallic or amphoric quality. Succussion is dependent upon the degree of distention with gas or fluids of the organ

at the time of distention. Gurgling sounds over the intestines are commonly noted and of extreme importance in the diagnosis. Dyspnea and cyanosis frequently result when the chest is compressed by lying on the healthy side. The abdomen has a tendency to be retracted, but extreme degrees of meteorism may be present when there is incarceration. Splenic dulness is present or absent, depending upon the location of the spleen; the same is true of the liver. Subjectively, eating and drinking often results in the sensation of fulness and oppression. In some cases the dislocation of the stomach results in a kinking of the esophagus causing difficulty in the swallowing of food. This is often more marked with the taking of liquids than with semisolids and solid foods. For the same reason it may be impossible to enter the stomach with a bougie. The presence of large intestines distended with air or fluids in the chest cavity frequently results in changes in physical findings similar to those following the ingestion of fluids by mouth. Constipation is commonly complained of, although it may not be present constantly.

Roentgen Ray Examination.—By far the most important diagnostic measure is flouroscope and plate examination. Through the use of these methods a positive diagnosis can almost invariably be made. However, the exact contents of the hernial cavity may not be determined so readily. In order to complete the diagnosis the patient should be examined at different intervals, and positions with and without the use of opaque fluids by mouth and per rectum.

Differential Diagnosis.—Although the differentiation between eventration, true and false hernia, is paramount in considering the prognosis and possible treatment, in many instances the diagnosis offers many difficulties. When the visible movements of the diaphragm or even the outline of its shadow can be noted we have a very considerable aid in the diagnosis. The relationship of the diaphragm to the foreign content of the pleural cavity is of importance. Vogel speaks of an inspiratory elevation of the prolapsed viscera in the presence of large defects in the diaphragm, while in eventration the flouroscope shows an ap-

pearance approaching the normal inspiratory descent of the diaphragm. Other conditions which must be differentiated are esophageal stenosis with dilatation, either acquired or congenital, esophageal diverticula, pathologic conditions of the mediastinum, pneumothorax, subphrenic abscess, enlargements of the liver and spleen, and other intra-abdominal conditions, resulting in an upward displacement of the abdominal organs with or without eventration of the diaphragm.

Prognosis.—Three main elements which enter into a consideration of the prognosis are: The type and size of the hernia, the degree of interference with the normal development of the individual, and the possibilities for surgical interference.

In eventration of extreme degree in the newborn there is usually an early fatal ending. This is even more true of the true and false hernias. In infants surviving their first year moderate eventration is compatible with good physical development. In those cases of extreme degree and in the hernias the prognosis must be based on the physical condition of the individual infant and the tendency toward the development of complications. In the cases under my personal observation the cardiorespiratory complications have been far more common, with a tendency to recur or become progressively worse, than have those of the gastro-intestinal tract.

Treatment.—The limited number of cases of diaphragmatic hernias recorded among the newborn must lead to the conclusion that premature expulsion of the fetus is a frequent result. Little is to be hoped for through operative procedure in young infants. In older infants and children with hernias of moderate size and in accessible locations operative interference is to be considered. Even in these cases the child must show at least a fairly normal development. The heart and lungs must give evidence of an ability to carry on the necessary physiologic functions. A careful roentgenologic study of the lungs will usually give information of value as to the condition of the lungs on the involved side as well as ability to respond to the requirements of the child on the part of the opposite lung. In many of the massive hernias there is almost complete atrophy

of the lung on the same side, and it must be remembered that the emptying of the chest cavity of its abdominal invasion leaves an empty space which must be filled with either the chest organs on the opposite side or it will have a tendency to result in eventration from below unless the chest wall itself is sufficiently elastic to permit it to collapse.

An emergency operation may be necessitated through torsion of the esophagus, a gastric crisis, or intestinal obstruction. The history of most cases diagnosed at operation, as shown by the recorded literature, shows this condition to have been mistaken for an intra-abdominal obstruction, the true condition not being diagnosed before the time of surgical interference. Most surgeons are agreed that the best results are to be expected from operations above the diaphragm. Richter recommends a long intercostal incision made at about the level of the sixth or seventh interspace, with resection of one or more ribs, in most instances at least the seventh.

Such an incision gives free access to the thoracic surface of the diaphragm, and the entrance of air facilitates the reduction of the hernia. The further steps recommended by Richter are closure and division of the sac at the diaphragmatic opening, which is followed by suture of the ring. He further suggests that when the peripheral portion of the opening is close to the thoracic wall, leaving no roon for suture, the median margin of the opening should, if possible, be sutured directly to the chest wall. In emergency operations, where the abdomen has been opened through mistaken diagnosis, an attempt should be made to close the diaphragmatic opening from below. Recurrence of the hernia may be hampered by suturing of the stomach to the margins of the ring and further anchoring to the anterior abdominal wall. In most instances it is impossible to remove the hernial sac when the operation is performed through the abdominal cavity.

Case I.—Baby G. Eventration of the Diaphragm (Fig. 120).—I am showing the radiograms of this case, and will speak of the postmortem findings. Baby G. was born in the maternity depart-

ment of Michael Reese Hospital in the service of Dr. Ludwig Simon. Report of this case was made by Dr. Irving F. Stein. This is a case of eventration of the diaphragm with the stomach, the greater part of the large and small intestines, the spleen, the left lobe of the liver, the tail of the pancreas, and the upper pole of the left kidney in the left chest. The heart was greatly enlarged and displaced to the right. The lungs were found above and to the right of the diaphragm, all compressed except the ower lobe of the right lung. The diaphragm on the left side was

Fig. 120.—Case I. Age twenty-six days. Eventration of left diaphragm. Stomach filled with bismuth meal. See autopsy report.

found to be a fibrous dome, thin and gray in appearance, extending to the left of the second interspace.

History.—At birth a marked asphyxia livida was present, and no effort at respiration was made until artificial measures were employed for twenty minutes. Cyanosis did not then entirely disappear and the pulse and respiration were very rapid. Examination at this time revealed a dextrocardia, scaphoid belly, and undescended testes. The birth weight was 3310 gm. Great difficulty was encountered in nursing, with marked cyanosis

and convulsive movements when at the breast. The infant was taken from the breast and given mother's milk by bottle. Respirations continued very rapid, and spells of very marked cyanosis occurred. Physical examination revealed the left chest tympanitic, the whole abdomen dull, and the edge of the liver not palpable. The belly was markedly retracted. The bow-line of the chest was flaring. Gurgling sounds were heard in the left chest, and breath sounds were heard only in the right chest posteriorly. The heart-beat was rapid, but the tones were clear.

The course during the first twenty-five days of life was marked by repeated attacks of very deep cyanosis, during which the infant's condition became desperate. On the twenty-sixth day the child died during one of these cyanotic attacks.

Roentgenologic Examination.—Figure 120, taken on the fourth day of life three hours after the ingestion of a bismuth meal, shows the stomach and intestines in the chest. It is impossible to say which intestines are present from this plate. Another plate was taken after death, with bismuth injected into the bronchi. Only the lower lobe of the right lung admitted the bismuth emulsion. The gas distention of the stomach and bowels here beautifully portray the extent of the eventration.

Case II.—John E. W., Aged Six and a Half Years. True Diaphragmatic Hernia (Figs. 121–123).—This boy is a second case of true left diaphragmatic hernia with dextrocardia. The hernial sac contains practically the whole of the stomach, a small amount of small intestine, and a large part of the colon. The hernial sac extends as high as the second rib anteriorly.

History.—The patient first came under my observation at Sarah Morris Hospital May 7, 1919, and remained under observation for twelve days. He had always been an irritable infant. His sleep was broken, awakening frequently with a sharp cry as if in pain. He remained retarded in development and has always been below the normal weight for his age. He entered the hospital for a cough and moderate temperature, with which he had been ill for one week.

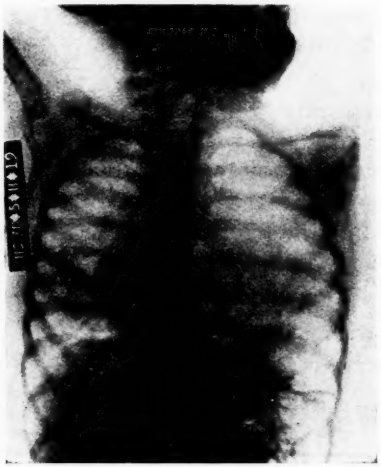


Fig. 121.—Case II. Age three years. True left diaphragmatic hernia. Radiogram on admission.

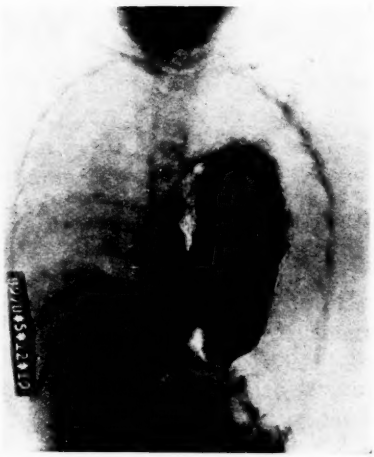


Fig. 122.—Case II. True left diaphragmatic hernia. Stomach filled by bismuth meal.

Past History.—Spontaneous delivery. Was still partially breast fed when first seen at eighteen months, at which time he

was also receiving milk, cereals, and vegetables. He suffered from a chronic constipation, but of only moderate degree, which occasionally required laxatives.

Past Illnesses.—He has had repeated slight illnesses associated with fever and numerous attacks of moderate bronchitis. The only gastro-intestinal tract disturbances other than constipation with which he was ill during his first eighteen months was at four months, at which time he had a severe vomiting spell, which, however, ceased spontaneously.

Fig. 123.—Case 11. True left diaphragmatic hernia. Colon filled by bismuth meal.

Physical Examination.—The patient is a poorly nourished, underdeveloped white male child, who shows no outward signs of acute respiratory or gastro-intestinal disturbances. The skin has a dull hue which, however, cannot be described as truly cyanotic.

Weight 32 pounds. Temperature 98.6° F. Pulse 124. Respiration 24. Scalp, ears, and eyes are negative. The nose is poorly developed and shows partially bilateral obstruction. He is a mouth-breather, and on palpation there is a considerable

increase in adenoid tissue found. His tonsils are small, his tongue coated. Neck is negative except for a slight adenopathy. Chest is asymmetric, with a well-developed right side, with good excursion, while the left side is flat, and there is practically no movement with respiration. Over the right lung there is moderately increased tactile fremitus, hyperresonance, prolonged and harsh experium, with numerous moist râles heard throughout the posterior half of the lung. These findings suggest a moderate bronchitis with a possible emphysema. The left chest is poorly developed, there is flatness in the lower fourth, with tympany above, and dulness over the apex. Breath sounds are completely absent except at the apex of the chest, where breath sounds are heard, but they are distant, and over this region and in the axilla numerous moist râles are heard. There is an absence of tactile fremitus over practically the whole of the left chest. Upon questioning the patient we find that he has been fed recently, and that he consumed about 16 ounces of fluids and semisolids, which, in all probability, accounts for the flatness in the lower chest. There is complete dextrocardia, heart sounds are normal. The abdomen is soft and moderately distended. No abnormalities are noted, the liver being in its normal position and the splenic area of dulness being present. Genitalia, extremities, and reflexes are negative. Urine is negative. Blood, hemoglobin 55 per cent., white blood-cells 11,000, leukocytes 55 per cent., Von Pirquet is negative, feces negative.

Roentgenologic Examination.—The plates in this case, taken before and after an opaque meal and enema, confirm the diagnosis of diaphragmatic hernia, seemingly of the true type, with the stomach revealed almost in its entirety in the left chest, extending almost as high as the second rib anteriorly. A few loops of small intestine are noted and a large part of the colon. The heart is displaced to the right chest.

Summary.—This case is of especial interest because the diagnosis remained unsuspected throughout the first eighteen months of life. Other than for a retardation in its general development its illnesses were limited to one attack, described as

stomach trouble at four months, and a moderate attack of bronchitis. The diagnosis was of a true condition existing in the chest made through roentgenologic examination after observing bilateral chest findings which were difficult to interpret in the infant, which was, to all appearances, in a fair state of health.

Case III.—Elmer N., Aged Three Years (Figs. 124, 125).—This child has been diagnosed as one of *true left diaphragmatic*

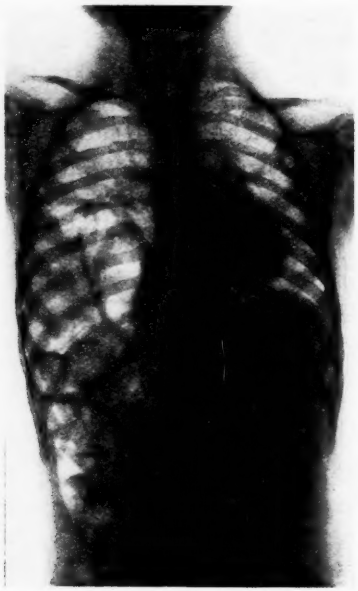


Fig. 124.—Case III. Age three years. True left diaphragmatic hernia. Radiogram on admission.

hernia and dextrocardia. The hernial sac contains the cardia of the stomach and the splenic flexure of the colon (Figs. 124, 125). He first entered the hospital at the age of eight and a half months (May 17, 1922).

History.—Normal birth at full term, breast fed to entrance. Past illnesses none. Family history negative.

Physical Examination.—When first seen a poorly nourished white male child apparently dehydrated and giving the appearance of having a respiratory involvement. Temperature 101° F. Respiration 50. Pulse 150. Weight 12 pounds. Scalp, eyes, ears, nose, mouth, and neck were all negative. Chest showed a well-developed rosary and Harrison's groove. Expansion was diminished on the left side and the left chest was poorly developed. Further examination showed dulness in the

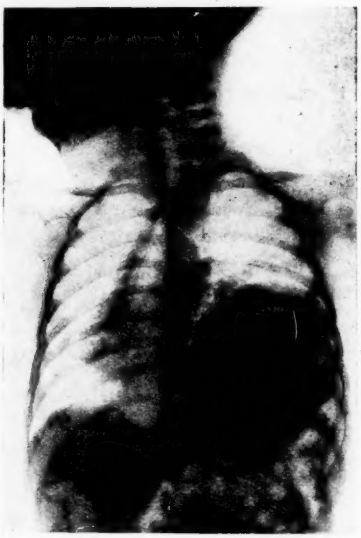


Fig. 125.—Case III. Age three years. True left diaphragmatic hernia. Stomach filled with bismuth meal.

right lower chest, with numerous moist râles and scattered areas of bronchovesicular breathing involving the whole of the right lower lobe. The lower half of the left chest was tympanitic and the left upper chest hyperresonant. Breath sounds were absent over the lower left chest. The diagnosis recorded was that of a right bronchopneumonia, with either a left pneumothorax of diaphragmatic eventration or hernia. The heart was displaced to the right with the apex-beat in the right fifth in-

terspace. The right border of the heart was 1 cm. external to the right midclavicular line. The heart sounds were not accentuated and no murmurs were heard. The abdomen was soft, flaccid, and there were no areas of tenderness; the liver was palpable ½ inch below the costal margin and the spleen was also palpable. The kidneys could not be felt. Genitalia negative, as were the extremities, except for evidences of rickets. Reflexes were normal.

Roentgenologic Examination.—Left diaphragmatic hernia with visible sac. The cardia of the stomach and the splenic flexure of the colon are in the chest. These findings were corroborated by a second examination following a bismuth meal.

Discharge.—Infant was discharged on the thirty-third day, having completely recovered from its acute illness, and was in good general health. The infant returned eight days later acutely ill, with vomiting and diarrhea as the most prominent symptoms. Discharged on the thirtieth day in fair general condition.

Course.—During the ten months since his discharge he had no acute illness. He has undergone a normal physical development.

Summary.—Breast-fed infant somewhat under weight, but who had never been acutely ill up to the time of the development of the present attack of pneumonia. Notwithstanding his undiagnosed congenital dextrocardia and eventration of the diaphragm, and the presence of the cardia of the stomach and splenic flexure of the stomach within the chest cavity, he has, to all appearances, suffered but little in his general development.

Further Course.—During the intervening two years there is a history of repeated attacks of generalized bronchitis and pneumonia.

Present Condition.—At three years we observe an underdeveloped child somewhat cyanotic—his weight is 22 pounds. Physical examination of his chest shows a well-developed right chest, normal tactile fremitus, hyperresonance, and somewhat exaggerated breath sounds on the right side. The left side of the chest is poorly developed, tympanitic in its lower half, and gives a fairly good note throughout the upper half. Breath

sounds are absent over the lower half; an occasional gurgling sound. Breath sounds diminished in character from the normal, with occasional râles, are heard over the left side. We will now feed the child in order to demonstrate the changed physical findings after filling the stomach. You will note that percussion in the upright position gives us a flat note to the level of the eighth rib posteriorly with a tympanitic note reaching as high as the level of the fourth rib posteriorly. Anteriorly there is less flatness. We now place him in the recumbent position, and you will note that the flat note over the lower chest has disappeared and is replaced by tympany. I wish to emphasize the importance of the relationship of food intake to the time of examination, and also the change of physical findings with changing position of the child.

The three following cases of *false diaphragmatic hernia* have come under my observation. Of the first 2 we present the specimens. The third case is of especial interest because the patient reached the age of thirty-one years without there being a suspicion of his chest pathology.

Case IV.—Baby J. S. (Fig. 126) with history as follows:

Family History.—Maternal: Mother aged twenty-eight years, in good health, pregnancy normal. Grandfather died of heart disease at fifty-five years; grandmother, of pneumonia, at sixty-two years. Paternal: Father living and well. One other child living and well, aged five years.

Personal History.—Child aged eight weeks when first referred to me by Dr. E. E. Simpson, giving a history of repeated cyanotic spells and short attacks of coma, some associated with vomiting, others not. The baby was well developed, but upon vigorous crying suffered from moderate cyanosis, associated with rapid cardiac action. Stools, except for constipation, were normal.

Physical Examination.—A provisional diagnosis of "situs inversus," with probable "congenital atelectasis of the left lung," was made because of the following findings: Displace-

ment of heart to the right, absence of breath sounds over the left lung, with area of dulness posteriorly and a tympanitic note over the entire left lower chest, abnormally large area of splenic dulness and the absence of normal liver dulness, with the palpation of a sharp notched edge of an organ pointing anteriorly and inward, and resembling the spleen in shape.

Fig. 126.—Case IV. Baby J. S., age six weeks. Congenital false diaphragmatic hernia. The left half of the diaphragm was displaced downward to the level of the navel and in its posterior outer half there was an opening about 4 cm. in diameter. The left pleural cavity was larger than the right and contained practically all of the small intestine, the ascending transverse and descending colon. The heart was displaced to the right and slightly downward. The stomach was in a vertical position, with the pyloric end extending into the pelvis.

Six weeks later, while visiting friends, the mother gave the baby, which had been breast fed, a bottle of cow's milk, shortly after which the child had a severe cyanotic spell, followed by coma, and death a few hours later.

Autopsy Report.—Well-nourished infant, presenting no external deformities. Upon opening the abdominal cavity a large dilated stomach immediately filled the incision. The stomach was in a vertical position, with the fundus and car-

dia upward, and the pyloric end extending into the pelvis, with the greater curvature anteriorly. To the left lay the enlarged spleen, and to the right the liver, also enlarged, with the lower edge pointing anteriorly and inward. The pancreas was also displaced with the stomach and lay along the lesser curvature against the vertebral column. The left half of the diaphragm was displaced downward to the level of the navel, and its posterior outer half contained an opening about 4 cm. in diameter, through which could be seen the upper end of the duodenum and the sigmoid, the only portions of the intestinal tract remaining within the abdominal cavity. The left pleural cavity was, therefore, greatly enlarged, containing practically all of the small intestine, the ascending transverse and descending colon, most of which were markedly distended; the left lung was about the size of a hen's egg; the right lung and pleural cavity were narrower, but probably not lessened in size because of the downward displacement of the liver and diaphragm on the right side. The heart was displaced to the right and slightly downward. The thymus was markedly enlarged and displaced to the right.

Case V.—Normal pregnancy, with spontaneous delivery of an eight-month infant, which lived six hours.

Autopsy Report.—Upon opening the abdomen the same was almost devoid of small intestine, an opening was found in the posterior half of the left side of the diaphragm, which was displaced downward, through which practically all of the jujenum, ileum, and the ascending and transverse colon had entered the left pleural cavity, resulting in complete atelectasis of the left lung, which was about the size of a walnut and confined to the region of the hilus of the bronchi on that side. The right lung was also only partially expanded. The stomach was displaced downward together with the liver and spleen, the former, as in Case IV, being almost in a vertical position.

Case VI.—Seen in the Allgemeinen Krankenhaus, Vienna.

Male adult, aged thirty-one years, entered the hospital for a right-sided pneumonia. Antemortem diagnosis: Right-sided

pneumonia, probably tubercular, with left-sided pneumothorax. His previous history so far as obtained was negative, except for repeated cyanosis upon exertion.

Autopsy Report.—A hernia was found in the posterior half of the left side of the diaphragm through which an ordinary sized fist could be passed. The left lung was poorly developed, displaced upward, and almost entirely atelectatic except for the apex of the upper lobe. The pleural cavity contained about 20 feet of small intestine, the other organs remaining within the abdominal cavity. No hernial sac was found.

In each of the 3 cases described the condition was undoubtedly congenital; they were also all of the false or ectopic type, as in each case there was an absence of a hernial sac. They were, as is the rule in non-traumatic cases, all left-sided, associated with complete atelectasis of the left lung, but in each case death was due to a different cause, namely, gastric and intestinal distention in the first patient, inability on the part of the right lung to carry on the respiratory function in the second, intercurrent disease (pneumonia) in the third case.

CLINIC OF DRS. JESSE R. GERSTLEY AND
L. J. WILHELMI

NORTHWESTERN UNIVERSITY MEDICAL SCHOOL

NON-OPERATIVE TREATMENT OF PYLORIC STENOSIS IN INFANCY

The Present Era May Be Called the Era of Physiology Rather than Pathology. Internists as Well as Surgeons are Realizing the Importance of an Estimation and Study of Function More than of Anatomy. The Whole is Greater than Its Parts. The Function of a Diseased Organ Must Be Improved Not Only for the Organ *per se*, But Also to Enable this Organ to Better the General Condition of the Body. The Reverse is Equally True, *i. e.*, an Improvement in the General Condition of the Body Causes a Marked Improvement in the Function of an Individual Organ. This Principle One of Us Has Attempted to Demonstrate in Previous Clinics. In the Treatment of Nutritional Diseases, Diarrheas, and Eczema in Infants He Has Advised Treatment Directed at the Body as a Whole Rather than the Individual Symptom. This Principle We Now Attempt to Apply in the Treatment of that Condition Known as Pyloric Stenosis.

Case I.—*History.*—This patient, referred by Dr. L. E. Frankenthal, Sr., was first seen at the age of five days. The father, previously in good health, was then suffering from acute articular rheumatism. The mother was slightly abnormal mentally; in fact, during her stay in the hospital she had delusions of persecution, thinking that the nurses were rather vicious in their attitude toward her and her child. She employed 5 different nurses in ten days. Other than this there was nothing in the family history. The labor was perfectly normal and uncomplicated.

When seen at the age of five days this child, who now appears to you as a perfectly healthy, fine, breast-fed baby two months of age, presented a rather emaciated, unhealthy appearance. The skin was quite jaundiced and around the eyes particularly pale and cyanotic. The main symptom, however, was vomiting with no apparent nausea. Practically nothing had been retained, nor—other than a little meconium the first day—had there been a bowel movement. The vomiting was projectile in type and occurred anywhere from immediately to several hours after nursing. The weight of the child at birth was 6 pounds, 8 ounces.

The clinical picture here suggested so definitely a case of atresia of some part of the intestine that an *x*-ray was ordered. This showed considerable retention of food in the stomach, but at the end of four hours 50 per cent. had passed through the pylorus and could be traced through the intestinal tract.

Case II.—This second child, two and a half months of age, and weighing 7 pounds, 8 ounces, seems to you in perfect health. When one month old he was brought to us with the following history:

The heredity showed nothing of importance. His birth weight was 6 pounds, 4 ounces. He was fed at the breast every three hours, and when this seemed insufficient was given a bottle containing 1 ounce of milk, 1 ounce of water, and ½ teaspoonful of sugar. Vomiting occurred, projectile in type, from the eighth day. It occurred immediately or some hours after feedings, and at times rolled out, at times was shot out. During these three weeks he had not gained and was hungry all the time.

Examination showed a poorly nourished, dehydrated child, with a wrinkled skin. In the abdomen no tumor masses were felt, but marked gastric peristalsis became visible following the drinking of a bottle of water. The *x*-ray showed that only 30 per cent. of the meal passed from the stomach in four hours, and that a diagnosis of pyloric stenosis was made by the radiographer.

These 2 cases serve to introduce the subject of pyloric stenosis in infancy. As you know, there are several prevailing ideas concerning the condition. One school claims that the condition is primarily and exclusively a hypertrophy of the circular and occasionally of the longitudinal muscles of the pylorus. There has been some dispute by pathologists as to whether there is a real hypertrophy in these cases or whether the apparent thickening of the musculature is due to the contraction found on operation or autopsy. That there is a definite hypertrophy, however, is shown by the recent work of Sauer, who proved his point by wax models. The second school believes the cause is a muscular hypertrophy with spasm associated, the hypertrophy being primary and the spasm secondary. The third school, advocated by as great a clinician as John Thomson, of Edinburgh, believes the spasm to be primary and the hypertrophy secondary.

Until pyloric stenosis can be produced experimentally it is difficult to decide which of these theories is correct. As far as clinical reasoning is concerned, however, we believe the latter view to be the most plausible, and the good results obtained from treating patients on the basis of such a theory is somewhat confirmatory evidence.

In favor of the spasm being primary is the fact:

I. That there is a relatively slow onset. Few patients develop symptoms before they are several days old. If the condition were a congenital anomaly it certainly would be present at birth.

II. Cases have been reported with findings in every way typical of pyloric stenosis in which the autopsy showed an absolutely normal pyloric musculature.

III. True organic stenosis—*i. e.*, a real atresia of the lumen of the intestines—rarely gives such a stormy symptomatology, and the patients, while showing an enormous dilatation of the stomach, do not present the same type of vomiting; in other words, in the type of case described by pediatricians under the terms of "pyloric stenosis" or "pylorospasm," there must be to some extent an associated gastrospasm.

IV. Finkelstein has described acute cases of this condition coming on at the age of one and a half months, running a stormy course for approximately ten days, and recovering.

V. No authentic autopsy on the newborn has shown a condition of congenital pyloric stenosis.

VI. That the condition must be largely due to spasm, one reasons from the fact that in almost every case where there is apparently a complete blockage some of the food at some time does pass through the pyloric orifice.

VII. Additional strength is given to this premise by the recent excellent study of Ballan, who has reported the pathologic examinations of 6 children dying after operation.

(*a*) Two of these children showed signs of some dilatation of the esophagus, suggesting an associated cardiospasm.

(*b*) The degree of hypertrophy of the musculature of the pyloric canal was not related to the age of the patient, but was related entirely to the length of time of the symptoms.

(*c*) This musculature showed in addition to the hypertrophy a marked increase in the amount and size of the elastic fibrous tissue.

(*d*) In one case of definite cardiospasm the elastic tissue was found markedly increased around the cardiac orifice and decreased around the pylorus. Here there was probably an atrophy of disuse.

VIII. Klee, by the stimulation of the vagus in decerebrated cats, has been able to produce the vomiting and gastric symptoms identical to those found in cases of so-called pyloric stenosis.

IX. Many of these cases show large amounts of mucus in the stomach. This, of course, could be explained on the basis of retention, but, inasmuch as the symptoms are always relieved by the removal of this mucus, also might indicate some anomaly of gastric secretion.

X. The very course of the disease is suggestive. The clinical symptoms rarely last over a few months, and after the disease has run its course the cure, whether surgical or dietetic, seems complete. Only a few cases show a recurrence of the symptom-

atology, though occasional autopsies, when the patient has died later from another ailment, may show some thickening of the pylorus.

The diagnosis as discussed in all texts is made by the classical symptoms of projectile vomiting (the vomitus at times showing signs of having remained in the stomach an abnormal length of time), by the peristaltic waves in the stomach region, by the constipation, by the tumor in the neighborhood of the pylorus (a method upon which I, personally, place little reliance), and by the *x*-ray examination showing a marked retention of the bismuth meal when the stomach should be emptied. By the amount of food retained in a given time investigators attempt to determine the degree of stenosis.

From my own point of view, however, the weakness of all this procedure is that it aims at anatomic diagnosis rather than physiologic. To the baby and to the baby's parents the nature of the local pathologic findings is of little import. The essential question is, "Does enough food pass the pylorus to permit the child to gain?" It has been my observation made repeatedly that all the symptoms (whether the anatomic diagnosis has been spasm or stenosis) *will disappear gradually, certainly, and in proportion to the gain in weight.*

A diagnosis made by *x*-ray would, according to this physiologic viewpoint, be of no great value except in those rare cases of absolute atresia. The fluoroscope shows only the conditions as they exist under that particular dietetic régime, but by no means shows what those conditions might be when influenced by other treatment or diet. Indeed, I have seen some cases where the feeding of the *x*-ray meal seemed to increase the vomiting and symptoms, and here the deductions from the *x*-ray would be entirely misleading.

In this same respect peristaltic waves mean very little. We have seen the most marked type of peristaltic waves in a condition which was obviously a spasm, and which disappeared very shortly under adequate treatment. Vomiting also is not a symptom to be treated *per se*, but a symptom to be considered only in connection with the general picture. Thus, if the baby

is gaining, the vomiting should be entirely neglected because it will disappear as the general condition of the child improves. No greater mistake would be made in a case of this sort than to change the diet or reduce it to stop the vomiting, and at the same time let the child's weight curve suffer. The worst cases one meets are those of children who have undergone a hunger cure for the treatment of this vomiting.

In short, I do not care about our present diagnostic signs and symptoms. If the baby can be made to gain he will unquestionably get well, no matter what the nature of the diagnosis. The whole is greater than its parts. Treatment should be directed first, foremost, and, above all, at the whole baby, and the best index of the whole baby is his weight and not any one particular symptom or sign.

Treatment.—The methods of treatment employed have been (*a*) surgical, as advocated by Fredet and later by Rammstedt; (*b*) medical, and (*c*) dietetic. The surgical treatment has been described so frequently that it is not necessary to mention it.

The medical treatment consists in the use of drugs which would tend to allay the spasm. Haas has suggested the use of large doses of atropin, and the Europeans have often gotten results from papaverin.

The dietetic treatment, first suggested by McClure for nervous vomiting, and introduced into the treatment of pyloric stenosis by Sauer, consists of the use of thick cereal. The formula for this cereal is as follows:

Skimmed milk,	9 ounces,
Water,	12 ounces,
Farina or rice flour,	6 tablespoons,
Dextrimaltose,	3 tablespoons,

Boil an hour or more in a covered double boiler until thick.

One can, of course, modify this paste as he sees fit.

Sauer advises particular attention to the consistency of the food. He believes that it should be so thick that when held on an inverted teaspoon it does not fall off.

Unquestionably one should bear in mind all these methods. A review of the literature must prove that there are cases which will recover only with operation. The use of drugs allaying spasm has certainly met with success. The idea of thick cereal has been most fortunate.

What I wish to suggest is not a new treatment, but the use of a certain principle. To my mind everything suggests a primary spasm of the musculature of the pylorus associated with a secondary hypertrophy. There is probably also a hypersensitiveness of the gastric musculature. The cause of the condition is unknown, possibly an as yet undescribed abnormality of gastric secretion. The indications for treatment then must be three:

1. To quiet and calm the child as a whole.
2. To relieve the local spasm as far as possible.
3. To get enough food through the pylorus, independent of all vomiting, *to permit the child to gain.*

I. As regards the first of these—*i. e.*, for calming the child as a whole—no agent can compare to a quiet unemotional environment and the care of a competent nurse.

It is remarkable to see how a child in one environment will seem unable to retain a single feeding, and in a quite different environment will be radically improved. Take for granted this baby (the child of the nervous mother). In the hospital the child was given small quantities of breast milk and retained it very well. As soon as the child was taken home it had a marked setback, vomited practically everything that was offered, lost weight, and became acutely ill. When taken from the mother and placed in a private room in the hospital in charge of a competent nurse and given small doses of milk expressed from a wet nurse, the child's recovery was perfect.

In addition, the use of a little bromid and paregoric may be of service.

II. As regards the second of these, *i. e.*, the relief of spasm, I have not met with success with any of the drugs advocated, though I have used atropin in large doses.

A method far more satisfactory occurred to me from the

following reasoning: If the condition is largely a spasm associated with a hypersensitive gastric musculature, perhaps in the period following muscular contraction, there might be sufficient muscular relaxation to permit food to pass even an organically hypertrophied pylorus. *This is just exactly what seems to happen.* You will be surprised to see in how many cases if you offer a baby his feeding, let him vomit, and then ten or fifteen minutes later let him take it again, how frequently he will retain it. Mucus also seems to accentuate the vomiting. I see no reason for irritating an entire throat, esophagus, and stomach and bringing in the factor of nausea by the persistent use of a stomach-tube. Let the baby wash his own stomach. If there is plenty of food let him wash the stomach with the food itself, thus giving him the benefit of any fraction of it that might be retained. In other words, combine stomach washing and refeeding in the same procedure. Fifteen minutes before the meal give the baby, depending on his age, ½ ounce or more of his feeding. Then, oblivious to any vomiting, give him the rest of the feeding in fifteen minutes. You will be surprised to see how often his second feeding stays down. If the supply of the food is limited, as with breast milk, I give ½ ounce of water for the preliminary feeding. The child at any rate gets the benefit of any water retained. It is advisable to have the feeding as hot as possible.

The success of this (one might say) local treatment depends, just as does the general treatment, upon the qualities of the nurse. A competent nurse of calm temperament soon learns the idiosyncrasies of the baby, and by the use of her common sense learns many little tricks which will enable the baby to retain a sufficient quantity of his feeding. One of these tricks is placing the child on his right side so that the food may run more rapidly through the pylorus during its periods of relaxation. But in every case the procedure may be different, depending upon the child and upon the intelligence and originality of the nurse. For this reason the treatment of a severe case in a hospital ward is almost out of the question, for no nurse is able to give the child the individual attention that he

needs, and no one nurse is with the baby long enough to learn the idiosyncrasies of that particular child.

The success of this refeeding technic suggested another little trick based upon the same hypothesis (*i. e.*, the sensitiveness of the gastric musculature seems to decrease during the period of feeding). We have tried successfully the following technic, giving the child a nipple with an extremely small hole for the first five minutes. It has to work hard during this period and does not get a great deal of food, and may regurgitate a slight amount; then as the sensitiveness of the stomach decreases a nipple with a larger hole is substituted.

III. As regards the third of these—*i. e.*, the absolute necessity of a rising weight curve—brings for consideration:

1. The nature of the food.
2. The number and size of the feedings.

As regards the nature of the food physicians have suggested breast milk, breast milk plus lactose, buttermilk and fat-free mixture, all kinds of conserves, and last, and best of all, the thick cereals.

Personally I do not think it makes a great deal of difference which you choose, if you *stick* to it. I believe the number and amount of the feedings to be of greater consequence, particularly if the above procedures are followed. Breast milk, of course, is the ideal from the standpoint of the baby's nutrition. Where the environment or nurse is not all that can be desired, unquestionably cereal mixtures give the best results. That these are not absolutely without danger is shown by a recent report in which 3 babies died of asphyxia or bronchopneumonia following aspiration of regurgitated thick mixture. In my own experience it seems that the cereal mixture need not be so thick as that usually employed. I have found oatmeal more satisfactory than farina. These, of course, are probably individual preferences.

Far more important than the nature is the number and size of the feedings. I believe *emphatically* in giving small quantities frequently during the first few weeks of the ailment. On such a schedule, even if the child vomits frequently, it will

retain more in twenty-four hours than upon a less frequent schedule. I start usually with ten or twelve feedings of ½ to 1 ounce, and increase gradually about ⅓ ounce in each feeding.

DISCUSSION

Chart I (pages 598, 599) shows the procedure and results in the first case described. When on two-hour feedings the child got about ten daily.

Chart II (pages 600, 601) shows the results in the second case, a child somewhat older. You notice that the child when put on breast milk, ten feedings of 1½ ounces, vomited continuously and was losing weight. I believe now I would have placed this child probably on ten feedings of only 1 ounce rather than 1½ ounces. Instead of making any marked change in the feedings, however, farina was added to the breast milk, with a resulting gain. Then, perfectly independent of the vomiting which you see scheduled at the bottom of the chart, and depending entirely upon the child's subjective sensations and *the nurse's observations and suggestions*, the feedings were gradually increased. It will be seen that though the vomiting continued, the child's weight curve went up, his general condition improved markedly, and after three weeks he was not vomiting at all, and discharged. In the Out-patient Department the cereal was gradually reduced and the diet brought to the one usual for his age. For the last two weeks he has taken a two-third milk mixture with dextrimaltose, seven feedings, and has been in the perfect health that you see now.

You may ask why we have shown the first patient. With only 50 per cent. retention even the surgeons would not have advised operation.

Isn't the fact significant that these 2 patients have had almost the identical symptomatology—one starting early, one a little later? Both symptomatology and *x*-ray suggested that the second case was more severe, the *x*-ray in the second diagnosing definitely a stenosis and recommending operation. Both cases made uneventful recoveries when treated according to the identical principle, namely, that the case was pri-

marily a spasm and would disappear with a rising weight curve.

But is there not another significance to these cases? Is it possible that Case I is just an early form of Case II, and that proper treatment right at the beginning prevented the development of more severe symptoms.

For ten years we have treated patients upon just such a principle. As soon as symptoms developed, active treatment was started, with the idea of establishing a hypothetic prophylaxis, and during these ten years in not one single patient was it necessary to resort to operation. Everyone made a perfectly definite and uninterrupted recovery with the exception of one possible case, and the relapse in this case was not absolutely definite. From such a point of view it would be unjust to say that no child should be operated. Naturally, if the condition has become well established, so that the degree of hypertrophy is far greater than the spasm, operation may become necessary. In our own experience we have never seen such a case, but we feel that this is due to the fact that we have always started active treatment as soon as any child shows the slightest symptom.

Let me emphasize again that the principle to be followed is the one quoted above, namely, that one is not trying to cure an anatomic condition, and that if the general condition of the child has been satisfactorily improved and the stomach and intestines normal as regards function, we are perfectly satisfied. It is possible that cases herein reported as cured if operated on for some other condition in after years may still show signs of a thickened pylorus, but as long as the stomach and intestines have been functioning normally and the child is in perfect condition, I see no more reason for having had such a child operated than there would be for removing in an adult a scar of a pulmonary tuberculosis that had healed in adolescence.

CHART I

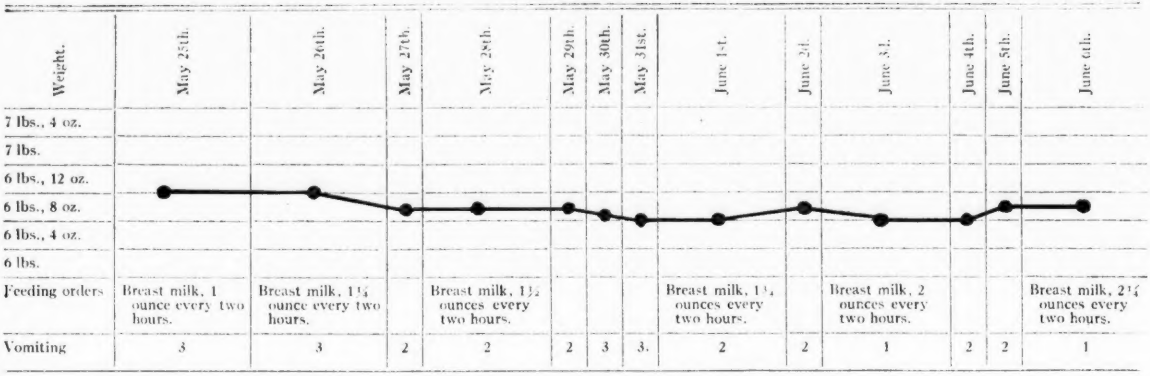
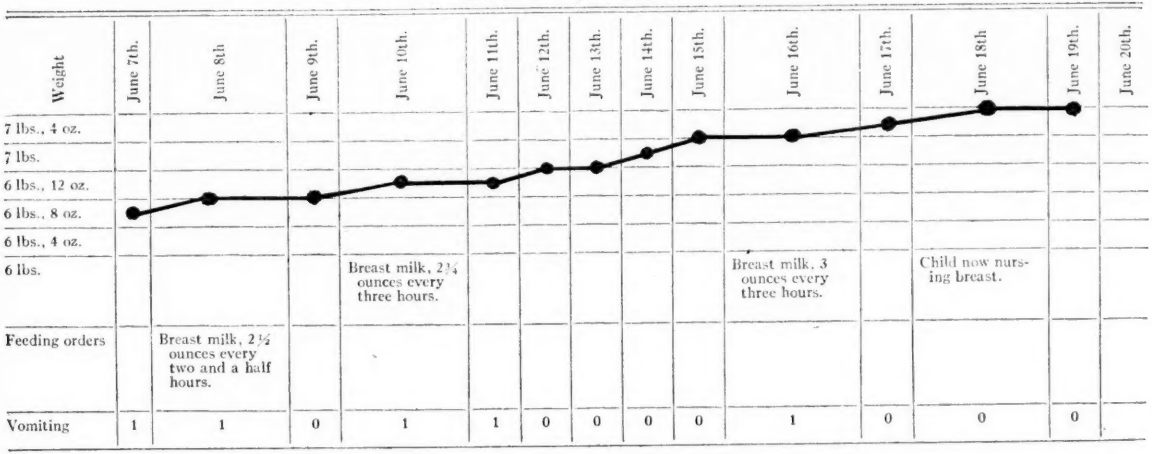

Weight.
May 25th.
May 26th.
May 27th.
May 28th.
May 29th.
May 30th.
May 31st.
June 1st.
June 2d.
June 3d.
June 4th.
June 5th.
June 6th.
7 lbs., 4 oz.
7 lbs.
6 lbs., 12 oz.
6 lbs., 8 oz.
6 lbs., 4 oz.
6 lbs.
Feeding orders
Breast milk, 1 ounce every two hours.
Breast milk, 1¼ ounce every two hours.
Breast milk, 1½ ounces every two hours.
Breast milk, 1¾ ounces every two hours.
Breast milk, 2 ounces every two hours.
Breast milk, 2¼ ounces every two hours.
Vomiting
3
3
2
2
2
3
3.
2
2
1
2
2
1

CHART I (Continued)

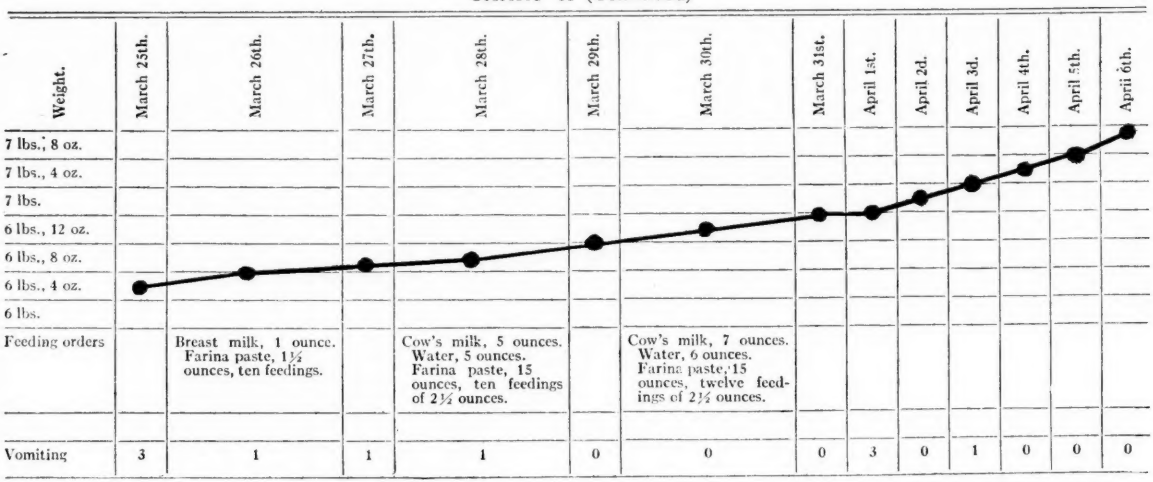


Weight	June 7th.	June 8th	June 9th.	June 10th.	June 11th.	June 12th.	June 13th.	June 14th.	June 15th.	June 16th.	June 17th.	June 18th	June 19th.	June 20th.
7 lbs., 4 oz.														
7 lbs.														
6 lbs., 12 oz.														
6 lbs., 8 oz.														
6 lbs., 4 oz.														
6 lbs.				Breast milk, 2¾ ounces every three hours.						Breast milk, 3 ounces every three hours.		Child now nursing breast.		
Feeding orders		Breast milk, 2½ ounces every two and a half hours.												
Vomiting	1	1	0	1	1	0	0	0	0	1	0	0	0	

CHART II

Weight.	March 14th.	March 15th.	March 16th.	March 17th.	March 18th.	March 19th.	March 20th.	March 21st.	March 22d.	March 23d.	March 24th.
7 lbs., 8 oz.											
7 lbs., 4 oz.											
7 lbs.											
6 lbs., 12 oz.											
6 lbs., 8 oz.											
6 lbs., 4 oz.											
6 lbs.											
Feeding orders	Breast milk, 1½ ounces, ten feedings.				Breast milk, 1½ ounces. Farina paste, ½ ounce, ten feedings.					Breast milk, 1½ ounces. Farina paste, 1 ounce, ten feedings.	Breast milk, 1½ ounces. Farina paste, 1 ounce, ten feedings.
		x-Ray taken.									
Vomiting	3	5	3	1	0	1	1	0	2	2	1

CHART II (Continued)



Weight.	March 25th.	March 26th.	March 27th.	March 28th.	March 29th.	March 30th.	March 31st.	April 1st.	April 2d.	April 3d.	April 4th.	April 5th.	April 6th.
7 lbs., 8 oz.													
7 lbs., 4 oz.													
7 lbs.													
6 lbs., 12 oz.													
6 lbs., 8 oz.													
6 lbs., 4 oz.													
6 lbs.													
Feeding orders		Breast milk, 1 ounce. Farina paste, 1½ ounces, ten feedings.		Cow's milk, 5 ounces. Water, 5 ounces. Farina paste, 15 ounces, ten feedings of 2½ ounces.		Cow's milk, 7 ounces. Water, 6 ounces. Farina paste, 15 ounces, twelve feedings of 2½ ounces.							
Vomiting	3	1	1	1	0	0	0	3	0	1	0	0	0

CONCLUSION

If one follows the principle that the whole is greater than its parts, that as the general condition of the child improves the local condition will improve proportionately, if one bears in mind the principle that the *x*-ray is an accessory in diagnosis and not a guide to treatment, if one aims his entire treatment at the general condition of the child rather than exclusively at the vomiting, a large number of patients will be cured who otherwise would necessarily have been operated. An additional aid to this treatment is the prophylactic idea that these cases are primarily a condition of spasm of the entire gastropyloric musculature with secondary hypertrophy of the musculature, and should be treated immediately with the onset of symptoms. Unquestionably there are cases which need to be operated and that will be operated. We believe, however, if one follows this sort of a prophylactic idea the number of patients requiring operation will be less and less.

CLINIC OF DR. CHARLES LOUIS MIX

MERCY HOSPITAL

CHRONIC NEPHRITIS

THE chief reason for showing this patient lies in the fact that he has been under observation for fourteen years. Somewhere I read in the writings of an English physician a notation to the effect that if we are going to learn very much more about some of our chronic diseases we must learn to study cases over long periods of years. This must be the work of older practising physicians, but it can be of value only when supported by records made at the time of observation. Fortunately in this case we have such records, and we are able to follow the evolution of a case of chronic nephritis over a period of fourteen years.

The patient was first seen on November 26, 1909. He was then thirty-four years of age. He complained that on September 1st he had what he called gastritis, evidently nothing but an attack of some sort of transitory indigestion, for it lasted but a few days. He was unable to give a fair account of it, but stated that it was followed by weakness, extreme nervousness, and insomnia, with a good deal of fear and anxiety. His reaction to his infection seemed to border upon an anxiety neurosis. He confessed that he had gradually gotten better, so that when I saw him at the end of November, fourteen years ago, he was feeling comparatively well. Further details of the attack of September 1st, which he was able to recall showed that the onset was very sudden at about 8 P. M. after a heavy supper and the smoking of two or three cigars. He went to bed, and at midnight felt suffocated by pressure against the heart. The doctor gave him something, the nature of which he does not know, and he began to vomit. His temperature was taken, and it was neither elevated nor depressed. Twelve days later he had a

similar attack, lasting a few hours, again associated with pressure against his heart, and again he became extremely nervous. The doctor who was called gave him two hypodermic injections. He does not know what they contained, but they were probably hypodermic injections of morphin, since the doctor who was called was in the habit of giving morphin to all of his acute patients.

My notes on November 26, 1909 record that his right brachial artery moved to and fro with each pulsation; that the arch of his aorta was palpable; that his aortic second tone was much too loud, but that the outline of his heart was approximately normal, there being no signs of cardiac hypertrophy at that time. It was felt that there was some relationship between the apparent rise of his aortic arch so that it was palpable in the suprasternal notch and the feeling of pressure in his thorax against his heart. No signs of gastric disturbance were then evident, the only findings being those of the arterial system itself. The urinalysis was entirely negative. Although the man was only thirty-four years of age he evidently was showing some signs of arterial disease. He was accordingly put upon Basham's mixture, 2 drams after each meal, and I gave him some emergency tablets of nitroglycerin for similar attacks, and suggested that he take nitrite of soda in doses of 1 grain after each meal.

I did not see him again until February 9, 1910. At that time he said that he had had no similar attacks of indigestion, but at times he had had a shooting pain instead of a constricting pain in his thorax. The same treatment was continued, and he was given some bromid of soda with strychnin.

On March 16, 1910 examination showed the same findings, with the added fact that the apex-beat of the heart was beginning to show lateral displacement. It was in the fifth interspace, but in the nipple line. He stated that he was on his way to West Baden for a week or ten days for a rest. Previous to this (February 12, 1910) I had a letter from the patient, which I quote as follows: "Since my visit to Chicago I have been worse in respect to my head, so much so that it makes me extremely nervous and very anxious as to the outcome. To describe the feeling,

I would say that it is not like an ordinary headache; not a sharp pain, but a sort of continued griping, a feeling of constriction or bearing-down pressure across the top of the head, affecting the temples and forehead. It has been with me in the afternoon and becomes worse toward bedtime. I woke up this morning about 7.30 after four or five hours sleep, with the same feeling in my head. At noon I felt a little sick to my stomach, with no desire to eat lunch. My old trouble has not been bothering me for the last week, and if you can prescribe something that will break up this trouble I will be very much gratified."

This is the first complaint I had from the patient as to what may be described as a disturbed metabolism. At the time it was quite impossible to come to any conclusion as to the cause of the headache which he complained of, but in the light of his subsequent developments it became very clear. Of course the early arterial disease made one suspect from the beginning the possibility of a very gradual oncoming nephritis.

I next saw the patient June 25, 1910. He said that he had been getting on indifferently well, sometimes better, sometimes worse in his subjective feelings, "Last week I felt fine, and yesterday I felt the trouble returning again." His weight was now 140 pounds, appetite was good; his sleep was somewhat disturbed. Sometimes he had trouble in his head, particularly the peculiar sensation on the top. It would go away for ten days at a time, and then recur. He had been taking bromid and strychnin. Last week he had pain in the arm and shoulder, followed by pain in the abdomen. Sometimes he had pain in the left wrist for half an hour at a time for several times a day. He had been attending to his business, but had not put into it the vigor he usually did. His tongue was slightly coated. His knee-jerks were present and normal. He did not complain of a bad taste in his mouth, but his tongue was not very clean. His blood-pressure taken on this day showed 128 mm. of Hg. systolic pressure and 81 mm. diastolic. The urinalysis was negative.

Seen again on July 21, 1910, he was complaining of pain in the back of his head which frequently is indicative of nephritic origin, and also pain in both parietal regions. He said he had a

good deal of headache, at times for a week, and pain in the right shoulder. Subjectively he felt weak. He thought that these pains were probably brought on by exposure to a rain storm. The pain in the back of his head was worse at night (as it frequently is in nephritic cases). For the last two days it has been better. He had one bad attack of disturbance with his stomach after a heavy meal. His feet felt as though his circulation was poor. The tongue showed slightly the imprints of his teeth. His bowels moved daily, but only by the influence of phenolphthalein. He could not take a hearty meal without having trouble, yet his appetite was good. His teeth, tonsils, and head sinuses were normal. No gall-bladder, appendix, or prostatic pathology was present. December 28, 1910 he consulted me again, and said that when he was busy he was all right, but always after supper he was nervous and unstrung and during the night he was invariably worse. During the summer he was out a good deal with his automobile. In the fall he had been inside a great deal more and had been somewhat confined to his office. His sleep was fairly good, but occasionally he had to take veronal in order to get his proper rest. The pains which used to bother him no longer disturbed him, though he still complained of obscure pains in the bowels and stomach. He had gained 6 pounds. His abdominal pain could be relieved by a whisky toddy, but not always. The heart findings were just the same, but his blood-pressure had risen slightly; it was 133/84 against 128/81. The urinalysis was negative.

On March 31, 1911 he complained of nervousness, especially toward evening, with tingling and numbness in the hands and feet and pains in the left shoulder and arm. He said that at times the *heart beat irregularly*, especially when he had pain. His appetite was good, but he could not eat a large meal. His blood-pressure had risen slightly, being 135 systolic and 92 diastolic.

A long gap now intervenes in my knowledge of this patient. I did not see him again until June 8, 1916. He told me that during these five years he had felt unusually well. He said he has been able to sleep and work, but lately he had been having

difficulty in sleeping. He was again worrying about his condition, though it was not so bad as in 1909. His weight was very much better. He complained of an ordinary cold lasting three weeks, of nervousness, irritability, and insomnia. He was dizzy and had pressure in his head and frequently put his hands to his temples. He perspired very easily at the time of his dizziness and head pressure. He had had some pain in his left arm. His heart was not bothering him. When he lay down his head hurt. He could not sleep without getting exhausted. For the last week or ten days especially, he had had pain in the left arm and side. During these five years he had had a hearty appetite and indulged it freely. When he felt bad he did not eat. His weight had increased from 140 to 165 pounds and he had weighed as much as 175 as against 140 pounds when I first saw him. His digestion was good, his bowels were fairly regular, but his blood-pressure had risen still higher. It was then 155 systolic and 105 diastolic. His heart and arteries were in precisely the same state, as far as I could determine, as they were in 1909. Apparently there had been no arterial extension of his disease. The urine examinations which we made showed no casts or albumin or signs of renal disturbances.

The patient evidently was not wholly satisfied with his interview with me, because I did not see him again until November 4, 1919. Again he came complaining of the same trouble that he had in 1909 and 1916. He had been engaged more and more in his business. He had been very successful, had worked very hard, had organized two or three factories in different parts of the country. His work became quite heavy between 1916 and 1919. Two years before, in 1917, he said he had somewhat of a nervous breakdown, that he wanted to have a good cry, but that he got on somehow until the summer of 1917, when he saw a physician in South Bend, Indiana, who said he worried too much about his business. From then until April, 1919 he was quite well. At that time he became dizzy and sick one noontime so that he could scarcely walk home. He felt weak and called in a physician, who said he was all right and gave him nothing, but from time to time after that he had had spells

of anxiety and uneasiness as to his condition. He does not think that his trouble has been due to overwork. During 1917 he also consulted a physician in Indianapolis, who said he thought his trouble was indigestion and imperfect elimination of waste matter. He then took up golf to some extent and had not been quite so inactive as formerly, though he still complained of subjective nervousness, indigestion, and dizziness. Whenever he was in a crowd he seemed to have more of this subjective dizziness, and when he was alone he was less dizzy. Occasionally he had sharp pains in the feet, hands, and back of his head. At times after business conferences he would leave exhausted, feeling as though he was "shot to pieces," with a disagreeable sensation in his head. A light meal made him feel better; a heavy meal made him feel much worse.

At this time he was sent to the hospital for some observation, and was examined quite thoroughly as to his stomach both by means of the Rehfuss test-meal and the fluoroscopic examination. Nothing definite was found in his stomach. The emulsion passed through the esophagus freely on November 22, 1919. The air bubble was normal, the stomach was of the hypertonic type, the pylorus was freely movable and pliable. Peristaltic function was rather active and free. The pylorus closed off squarely and freely to the tip. The duodenal cap formed perfectly and could be splendidly visualized. Radiograms taken at intervals of time showed the stomach to be wholly normal. A Wassermann test was made at this time and was entirely negative. He was dismissed from the hospital and wrote me after his return to his home, stating that he arrived home Sunday evening about 6 P. M. very much pleased and encouraged by the fact that I considered that further observations as to his stomach were not necessary. He said he retired about 10 o'clock, passed a most pleasant night, in that he went to sleep quickly, and did not awaken until 6 A. M., at which time he arose and felt just as good as one could feel physically and mentally. Yet in his letter he goes on to say that subsequently he began to feel uneasy, not from distress in his stomach, but from nervousness, "My circulation seems faulty below the knees and that queer

feeling comes over me. I retired at 10 o'clock; after reading the paper I must have fallen asleep. About 12.15 I awakened, finding my head covered with perspiration, while the rest of the body was cold and my throat was dry. I have had generally an unusual feeling over the top of the head and over the body which I could attribute only to circulation, as I had no pain in any other part of the body." Unfortunately I have no observation of his blood-pressure at the time he was in the hospital because we do not know where his hospital record is, but on December 14, 1919, a few weeks after he left the hospital, his blood-pressure was 144 systolic, the diastolic not being mentioned.

My next interview with the patient was on September 20, 1920, at which time he again spoke of being extremely nervous and apprehensive. He complained of trembling and of shaking in the knees and of a feeling of uncertainty in his feet. These feelings are not due at all to any disturbance of his spinal cord, because he has a perfectly normal cord. They are due entirely to the subjective feeling of weakness. His blood-pressure on this occasion had risen still further. It was now 180 systolic and 116 diastolic. He again complained of occasional indigestion, stating that he had seen a doctor, who gave him some calomel, which straightened him out. Always he complained of one of three things or all—stomach and indigestion, nervousness, and vertigo.

On November 8, 1920 his blood-pressure had fallen a little, being 172 systolic and 110 diastolic. Without going into deatils, his subjective complaints were pretty much as before, his cardiac findings meanwhile being much more evident, a hypertrophy of the left ventricle being quite easily made out. His aortic arch showed the same changes that it always had. His brachials were now fairly movable and the aortic second was quite sharp. On May 25, 1921 his weight was 162 pounds. His blood-pressure had risen still farther, 198 systolic and 124 diastolic. On June 16th his pressure had risen very slightly, being 200 systolic and 128 diastolic. At that time he complained of coated tongue, bad taste in his mouth, and disturbance of digestion. In taking

his blood-pressure for the first time there was evidence of a little weakness in his myocardium, in that there was inequality of his pulse, and although I recorded the systolic pressure as 200, it fluctuated between 194 and 200, some beats being strong and some weak. The Basham's mixture which he had taken for so many years was continued. On June 30th his blood-pressure had fallen somewhat to 190 systolic and 110 diastolic; the pulse-rate was 84. Though he had for the last year or so had to rise at night to urinate, of late he had not risen. He weighed 162½ pounds. The urinalysis was still negative.

On October 27th, four months later, he weighed 162 pounds, his blood-pressure was 198 systolic and 128 diastolic, and he was not rising to urinate at night. Again his urine showed no albumin or casts, no red blood-cells, and, so far as one could tell, it was a perfectly normal sample.

A year went by before I saw him again, the next interview being November 17, 1922. His weight was then 178 pounds, while the blood-pressure had increased to 216 mm. systolic and 126 diastolic. His pulse was 100 and it skipped a beat occasionally. He was complaining of pain in his head and in the back of his neck. Again he stated that he did not have to urinate at night and the bowels were moving fairly regularly. His tongue was coated. He was smoking 6 cigars a day and sometimes more. He was trying to cut down on his eating, but had a good appetite. The day previous to our interview he went on a trip and had no lunch. At night he was so hungry that he ate a very hearty meal which did not disturb him as much as previous hearty meals had done. He had been carrying on his business very strenuously, even spending as many as one hundred and twenty nights a year on Pullmans. He had also been elected to the legislature, and he hoped this would be a change for him, a sort of vacation. His teeth had been *x*-rayed and put in good condition; he had had some extracted. He still had pain in his head from one ear to the other, sometimes with blurring of vision. He said it felt as though there was a slight smoke coming over them. He had a tendency to rub them to clear up his vision. Examination by an ophthalmologist failed to disclose any retinal

trouble. He had a little feeling of cold in his chest and around his heart. He said that at times he had a little pain in his heart. On the previous Saturday night before I saw him he could not go to sleep because of it, and had to sit up and read for an hour. Urinary examination on this date for the first time disclosed a trace of albumin. We had examined his urine ten times during the preceding years and never had been able to find any trace of actual nephritis until November, 1922. No casts were present, not even hyaline casts, and no red blood-cells could be found.

The next interview took place early in April of the present year, at which time he had a blood-pressure of 218 systolic and 128 diastolic. He was definitely uremic, complaining of intense headache, or blurring of vision, with a foul, coated tongue, bad breath, but with no delirium and no convulsions. He was brought out of his condition by hot packs. Examination of his urine showed quite a fair amount of albumin, with a number of hyaline and granular casts. The urinalysis now definitely shows a distinct nephritis from the findings. His heart is now quite a bit enlarged. He has at the present time a relative mitral insufficiency notwithstanding the fact that his blood-pressure is as high as it is. The aortic arch is quite enlarged, lying high in the suprasternal notch He has a good deal of throbbing in the arteries of his neck, and his brachials are quite tortuous and move with each pulsation. He has a little arrhythmia and inequality of the pulse, and he is showing at the present time slight signs of cardiac defeat.

This case is shown because it contains observations of the evolution of what we call nephritis over a long period of time. The patient is now forty-eight years of age; he was thirty-four when first seen. On the first interview definite changes were made out in his arterial system which were suspected to be probably due to the fundamental underlying disturbances which we call cardiovascular renal disease. It often begins on the vascular side, and we do not have evidence of involvement of the heart and of the kidneys until after many years have passed. The early phases of the disease seem associated with metabolic

intoxication. This man's history is replete with heavy meals, great physical and mental labor, physical and mental strain. Undoubtedly he has always been deficient in his body chemistry, and instead of taking care of his food as a normal individual should he seems constantly to have manufactured by-products which have had an invidious effect upon his arteries, kidneys, and heart. For years the effect seemed to be wholly upon his arterial system. More gradually the blood-pressure rose year after year, until finally it has reached proportions which we recognize as those of true nephritis. His heart, which at first showed no hypertrophy, gradually showed more and more of it, and now it is not only hypertrophied but it is also temporarily dilated with a moderate mitral leak.

In connection with this case I am tempted to make a few general observations with regard to nephritis. In the first place, the kidney is not an organ of secretion, and no nerves whose function is to produce secretion have ever been found going into it. Our modern concept of the kidney is that it is an organ of filtration and resorption. The glomeruli, particularly Bowman's capsule, constitute the filtration apparatus and the tubules constitute the organs of resorption. Nature is wonderfully economical, and so, when Bowman's capsule filters through too much water and valuable basic salts, the tubules reabsorb such proportion of the water as is necessary, and they also reabsorb the valuable basic salts. The result is that the urine is always of a certain approximate specific gravity and of practically constant chemistry. When disease assails either of these parts of the kidney, trouble begins. If there be disease of the glomerular apparatus, if Bowman's capsule becomes clogged and unable to filter, we have a retention of body fluids, with a production of edema. If, on the other hand, the filter works all right, but the tubules are diseased, so that resorption does not take place, we have larger amounts of fluids than normal passing from the body—a condition of polyuria. There is, of course, in these latter cases no edema whatever. Depending upon the involvement of these two distinct parts of the body some clinicians have been content merely to describe kidneys as either

wet or dry, the wet kidney being the kidney of glomerular involvement, and the dry kidney being the kidney of tubular involvement; the term "wet" having reference to the associated edema, and the term "dry" having reference to the associated absence of it. These two generic terms, wet and dry, describe fairly accurately the broad outlines of kidney disease.

The cause of edema, however, does not lie wholly in the lack of liberation of fluids through Bowman's capsule. Not only are fluids not secreted, but toxic substances also remain behind in the blood-stream. There results sooner or later an acidosis which extends to the protoplasm of individual cells. Such acid protoplasm locks the water within itself, so that anasarca results. At times only those cells which are in the lowermost parts of the body become, by reason of the weakened circulation, so acidified; and hence in cardiac diseases only the feet and ankles are edematous. But in wet nephritis, where all the cells, wherever located, are thus acidified, anasarca, or the universal distribution of edema, results. Hence in the nephritic cases we commonly look for incipient edema in the eyelids, where the tendency for accumulation of fluid is greatest. In later cases we expect to find edema everywhere.

Depending upon whether the kidney is wet or dry, whether the pathology is glomerular or tubular, are the urinary findings. It is quite evident that if the glomeruli be generally involved albumin will escape through Bowman's capsule, and hence will be largely present; if the tubules be for a time the only portion involved, albumin will be absent. It is evident that red blood-cells will be a sign of glomerular nephritis, present in the wet kidney and absent in the dry kidney. Also it will be evident upon a bit of thought that the amount of urinary sediment does not necessarily bear any relationship whatever to the amount of trouble in the kidney. The amount of urinary sediment is merely an indication at the time of the examination of the current insufficiency of the kidney process. A very slowly developing case, such as the one just described, may for years show no urinary findings whatever, and yet we know that changes are taking place in the kidney all the time. For years a true

Bright's disease may be existing with a perfectly normal set of urinary findings. On the other hand, an exacerbation of the process may come along and load up the urine overnight with a large number of casts, albumin, red blood-cells, and white blood-cells. This is particularly true in acute cases. The acute case always shows an abundance of sediment, the chronic case often gives scarcely any sediment.

A few words in regard to renal etiology are perhaps advisable because they will give us a better understanding of renal symptomatology. Cases used to be described as acute and chronic. The concept of acute nephritis was a disease bilaterally symmetric, affecting both kidneys. This is not our present concept. We recognize acute pneumonia as patchily distributed in bronchial pneumonia or as affecting one lobe in lobar pneumonia. Since the lungs are easily gotten at in physical examination, the mistake was never made of calling pneumonia a diffuse process entirely affecting both lungs. Since the kidneys are not easily gotten at by a physical examination we have drifted into the faulty notion that in acute nephritis both kidneys are equally and wholly involved. This view is quite beyond reason. It is true that if the etiologic factor be a toxin uniformly distributed in the blood, there will be a uniform effect upon both kidneys. Yet it is equally evident that if the factor causing acute nephritis be bacteria, such bacteria may affect both kidneys, but possibly only one; and that if both kidneys are affected, they may be unequally affected. Some portions of the cortex may be very much involved and other portions of the cortex very slightly involved. So it happens in acute nephritis that at times a great abundance of sediment may appear in the urine because of the great intensity of a very small focus of disease in one or both kidneys, and yet there is so much normal kidney not involved that elimination of urine goes on perfectly well. We know that this is true from our studies of blood chemistry. Some cases of acute nephritis with very intense findings in the urine exist with very slight reduction in the retention of creatinin, urea, and urea nitrogen in the blood-stream. Indeed, it has now become axiomatic that the proper way to arrive at a

conclusion as to the degree of renal involvement is not to measure the urinary sediment as disclosed by examination of the urine, but rather to estimate the residue in the blood-stream of the toxic substances which should be eliminated; and consequently no case of kidney disease is completely investigated until a chemical blood analysis has been made. We repeatedly find, for example, large amounts of creatinin, urea, and urea nitrogen in the blood-stream, with very slight findings on the part of the urine, or normal amounts of these substances, with extensive urinary sediment. It is perfectly clear, therefore, that the weight of the examination should be upon the blood retention of waste products rather than purely upon the elimination of waste products in the urine itself; in other words, instead of determining the urea in the urine we should determine the urea in the blood.

Of course, if the disease of the kidneys is caused by toxins of bacteria or chemical or metabolic poisons, then there will be bilateral disease of both kidneys and approximately equal distribution of the pathologic consequences. Bacterial intoxications are, as a rule, acute and glomerular; for instance, those following tonsillitis, diphtheria, and scarlet fever are almost invariably glomerular, associated with edema and large amounts of urinary sediment, plenty of albumin, and red blood-cells. So also is the urine caused by intoxication from the bile, a urine with abundant sediment because of the effect of bile upon the glomeruli and tubules. On the other hand, as in the case which we have just related, gastro-intestinal toxins appearing in the blood over a period of fourteen years in comparatively small amounts too slight to measure produced changes in the kidney so insidious that the urine for years was without sediment. These metabolic factors apparently affect the glomeruli scarcely at all. Their chief effects seem to be upon the tubules, and they give rise to what used to be called chronic interstitial nephritis, but which we are now rather fond of calling "chronic tubular nephritis." It is this sort of toxin which causes cardiovascular-renal disease. Bacterial toxins cause an uncomplicated disease of the kidneys, generally of the glomeruli, but these metabolic gastro-intestinal

toxins cause cardiovascular-renal disease. Hence we find, as in the case just described, a beginning effect upon the arterial system. There were early signs of arterial strain, of arteriosclerosis, so that at the age of thirty-four in this man there were found brachial arteries moving laterally with each pulsation. The aortic arch may be found to be high in the suprasternal notch and the aortic second actively accentuated, with an hypertrophy of the left ventricle. As time goes on extension of the effect of the toxins on the tubules of the kidney is noted, and it is not until after many years have elapsed that one can get definite signs of further extension of the disease in the kidneys. Then we find not infrequently that what was a tubular nephritis, involving only the basal portion of the tube, may begin to affect the convoluted tubules, and then Bowman's capsule, and so albumin begins to appear in the urine in addition to the earlier appearing hyaline and granular casts.

In general, we may say at the present time that we can fairly readily distinguish wet and dry kidneys. The wet kidney, or the kidney of parenchymatous nephritis, is of the glomerular type, and we can not infrequently note a case which may well be described as glomerulotubular, in which the process begins with the glomeruli and extends to the tubules—the old diffuse nephritis. The dry kidney, or the kidney of chronic interstitial nephritis, is of the tubular type, and we are able at times to distinguish in the later development of these cases a variety of nephritis which we may call tubuloglomerular in which there is an extension upward to the cortex from the tubule by the intoxication degeneration. Thus again we have another variety of the old well-known diffuse nephritis.

At the present time the proper way to look upon renal disease is from the broad point of view as to the character of the toxins and their effect upon the excretory or resorption apparatus of the kidney. We are to have in mind the possibilities and extent of kidney involvement which will be determined largely by the retention products in the blood. We are not to pay too much attention to urinary sediment. We are to pay more attention to cardiovascular involvement. By having our attention directed

to the broad aspects of nephritis we shall best be able to arrive at a much keener conclusion as to individual conditions.

It is interesting to note the manner of death in these various cases. The acute cases, the glomerular cases, are apt to terminate by uremia by retention of products which should be eliminated. On the other hand, the interstitial type, the tubular type of case, is apt to terminate by heart defeat, just as we have seen in the case we have described, where a gradual rise of blood-pressure from 128 systolic to 218 systolic is greatly burdening the heart. This patient is now beginning to show signs of actual cardiac distress. He has occasional intermittency, some inequality, and today he is beginning to show mitral strain. This defeat of the heart probably will be rather slow in evolving, and may take five or six years more before its completion, but I would not be surprised if this patient did not pass his fifty-sixth year. He is now forty-seven; it takes possibly twenty years or more for the evolution of one of these cases to its pathologic maturity. It is such an insidious process. It seems very difficult to check it in any way, and yet one feels after following a patient's career for fourteen years and watching the gradual evolution of the nephritis that if the patient had only been willing to accept some of our suggestions that his process of pathologic decay would have been very much delayed. We never could get him to curb his appetite. His weight went from 140 to 175 pounds against our strenuous advice. He indulged in hearty meals again and again when we told him he should not. In competition with business men he would eat a business man's lunch, consisting chiefly of a large steak, at some of his conferences, when we begged him to be temperate in his eating. He spent at one time a great deal of time in Pullman sleepers when he should have been sleeping in bed. He worked altogether too hard in his organization work and in his rather large business affairs, with the result that he increased the formation of his metabolic toxins and so hastened the evolution of his disease.

When one encounters a young man, thirty-four years of age, with signs of arterial trouble and with vague complaints of heart trouble and nervous trouble, disturbances of circulation, and,

above all, with frequent digestive upsets, one should have in mind the ultimate possibilities of such a case. We should implore our patients to be careful as to their present conduct in order to assure their future welfare. I feel certain that if this individual had been more temperate in eating and activity that we should not be at the present time looking a bad prognosis in the face; for we fear that he ultimately will die of heart defeat. We think that his blood-pressure, which is necessarily high in order to admit of filtration through the glomeruli, will remain high. We know that the heart will be able to carry on for a time, but ultimately it will not. It is not likely that he will show an out-and-out blockade of his glomeruli at any time, although he does now show signs of a mild uremic intoxication. His blood-vessels may not be in very good condition, and the possibility of a rupture of the cerebral blood-vessels is always present in such a case, but where one blood-vessel ruptures at least four hearts will suffer defeat in the disease; and the chances are that a stroke will not be the fate of this patient, but rather a heart defeat.

We cannot brag much as to treatment. We do not know any method whereby at the present time these toxins from the alimentary canal can be destroyed. I have always felt that they were capable of being split up by iron, and I have a reason for that belief in the efficacy of Basham's mixture in these cases. This man has been taking it off and on for fourteen years with a success that is perhaps indifferent, and yet we do not know but that his present state might have been much worse had he not been on this treatment. Better by far than destroying the toxins, however, is avoiding their formation. If only these individuals will be careful as to their habits of life and activity they surely will live very much longer.

CARDIOSPASM

THE patient is a married woman fifty years of age. She enters the hospital complaining that her stomach bothers her. On questioning it is evident that the trouble is regurgitation of her food which takes place with little or no nausea. She says that when she eats, the food goes down a short distance, but apparently not all of the way, and that usually she is obliged to spit up the contents. She declares that the contents come up sour, which would indicate that they reach the stomach, but on cross-examination she is not quite certain whether the contents are always sour. She used to weigh 160 pounds and now weighs 113. She attributes her illness to influenza which she had about a year ago. Following it she was left in what she describes as a very nervous state, and, as we shall see later, this was the real etiologic factor. She has no pain except at times after the regurgitation of food. She has a feeling as though all of her organs were asleep. At times her stomach bothers her so that she induces vomiting. Notwithstanding her distress she says her appetite is good, but she is afraid to eat. She is quite constipated. She has been to a hospital here in the city where her stomach was *x*-rayed, but there has never been any examination of the stomach contents following a test-meal.

The report of the *x*-ray which was then made was as follows: "Aorta normal; heart slightly enlarged, the greater enlargement being about the left side. Muscle tone somewhat weakened, but no definite signs of pathology are shown. Hilus shadows and a few calcified glands, with normal density of the hilus on both sides. The lungs, pleuræ, and bronchi are normal. Respiratory excursion is good. *Mediastinum:* This appears to be filled up in the lower three-fourths with fluid. *Esophagus:* Upon administering a thin barium mixture a constriction was found at the lower end of the esophagus at about the point where it passes through the diaphragm which has almost completely closed the opening into the stomach. The fluid, even as

thin as milk, will remain in the esophagus and not pass into the stomach for an indefinite period of time. No filling defect suggestive of a carcinoma is present at this time. The fluid appearing in the mediastinum is apparently water taken several hours previous to this examination. After administering 1/50 grain of atropin this constriction does not relax, the same thin mixture remaining in the esophagus as before. Examining the patient one hour after administering a small quantity of thin meal shows practically none of the food in the stomach. It is almost impossible to say definitely that this is not a malignancy."

Her family history was negative as to carcinoma. A brother and sister are living and well. A sister died from some infection. Her mother is living, but had a stroke recently. Her father is dead from pneumonia.

The patient, notwithstanding her history of trouble with the esophagus and despite the great loss of weight, was given a test-meal which was removed by the Rehfuss tube. We were emboldened to do this because the blood examination showed a hemoglobin of 88 per cent., red cells 5,320,000, white cells 5200, and the differential white count showed nothing abnormal. We felt that so high a percentage of hemoglobin and so large a number of red blood-cells could not possibly be associated with carcinoma of the esophagus, and we felt it probable that the disturbance was a cardiospasm rather than a malignant stricture, notwithstanding the adverse report which she brought with her. We succeeded in passing the Rehfuss tube without a great deal of difficulty, and seven samples were removed in the course of three and a half hours. These samples showed a total acidity of 14, 12, 14, 16, 16, 20, and 18 respectively, but there was no hydrochloric acid in any of the seven specimens. Lactic acid was present and a Gram-positive bacillus resembling the Oppler-Boas bacillus was found. Notwithstanding the presence of lactic acid, the absence of free hydrochloric acid, and the suspected bacillus, we still were very much of the opinion that her trouble was not a carcinoma either of the stomach or of the cardiac end of the esophagus. (It sometimes happens that malignant tumors occur on the gastric side of the cardiac orifice

and that they grow up into the lumen of the esophagus, behaving like carcinomata of the esophagus, but really being carcinomata of the stomach and giving rise to the usual laboratory findings.)

We made a fluoroscopic examination of the patient and, aside from the moderate enlargement of the heart, found nothing wrong in the thorax. Fluoroscopic examination by means of a barium meal showed very marked dilatation of the esophagus due to a constriction of some sort at the lower end near the cardiac end of the stomach. However, during the process of the examination, the emulsion passed through the constriction rather steadily, but in a small stream. The fluoroscopic examination of the stomach showed it to be of the atonic type and freely movable. Peristaltic function was active and the pylorus closed squarely to the tip. A well-formed duodenal cap could be visualized without manipulation.

As a result of these findings it seemed likely that the patient was really suffering from a spastic contraction of the cardiac orifice of the stomach. This is ordinarily called a cardiospasm and has usually been treated by forcible dilatation.

TREATMENT

Cardiospasm occurs in nervous individuals and really has to be treated by a process which will overcome the nervous apprehensions which these patients have when they attempt to swallow. At this point a long digression is necessary.

Unstriped muscle-fiber in the human body is under the control of the sympathetic nervous system, which now is more properly called the autonomic nervous system, and which really works in a sense automatically. This unstriped muscle-fiber is widely distributed throughout the length of the alimentary canal and is also found in the various ducts, in the uterus, and bladder. Whenever a defect takes place in the territory of supply of the sympathetic nervous system, reflex disturbances may occur. Perversions of afferent impulses to the sympathetic nervous system may give rise to reflex perversions; for example, disturbances on the part of the ovary very frequently lead to spastic contractions of the uterine muscle.

More important perhaps, especially in the consideration of this very case, than these anatomic causes for perversion of reflex action is the influence of the emotional state upon the sympathetic nervous system. When a person is in a state of emotional excitation a perverted set of reflexes, as a rule, are sent forth through the sympathetic nervous system. Anatomists have not succeeded in pointing out the pathway of such reflexes, but that it exists cannot be doubted. For example, in great grief the function of the stomach ceases and even hunger stops, probably because of the inhibition of the activity of the sympathetic nervous system because of the emotional state. The possibility is that the emotional center is located in the *regio subthalamica*. This is the *terra incognita* of the central nervous system. That it has an important function can admit of no doubt; that it is connected with the spinal cord in some curious way, even with voluntary innervation, is shown by the fact that in rare instances hemiplegics in yawning occasionally will stretch both arms, and yet when they voluntarily attempt to move the paralyzed arm they find they are powerless to do so. Thus it would seem that the innervation of these pyramidal tracts can sometimes be instituted at a lower level than the idealization cortex in the cerebral hemispheres. Similarly, from an *a priori* consideration, there must be some relationship between the emotional center, wherever it may be located, and the fibers to the unstriped muscles presumably under the control of the central nervous system. For example, in great fear it is a familiar fact that the salivary secretion will sometimes be completely inhibited, for people are said "to spit cotton." Fear in lower animals frequently leads to spontaneous evacuation of the contents of the bladder. The reflex is not a reflex which comes from any connection with the psychic centers. It is, instead, a reflex through the emotions. The ancient biblical saying that King David's "bowels were moved with compassion" is another expression of this relationship between emotion and unstriped muscle-fiber.

Bearing upon the location of this center in the *regio subthalamica* is an interesting experiment upon pigeons whose cerebral hemispheres have been removed. I am told that pricking

of the subthalamic region in such pigeons stimulates the erectors of the feathers. The phenomenon of blushing and of gooseflesh in the human organism can be explained only upon the basis of a primary disturbance of an emotional center, and the subsequent delivery of efferent impulses through the sympathetic nervous system to the periphery of the body.

Still another example of the influence of an emotional center in sending out perverted efferent impulses is afforded by exophthalmic goiter and hyperthryoidism. It has long been taught that an etiologic factor, of course not the sole one, is psychic trauma. But the term "psychic trauma" is not strictly correct. It is an emotional trauma, and, naturally, such an emotional trauma comes through the mind. It requires an emotional upset to send down perverted impulses through some pathway in the spinal cord to reach the autonomic system; and when such upset does send down these perverted impulses to the thyroid gland alone, it is stimulated to the excessive formation of thyroxin and the well-known intoxication signs develop.

The anatomists are unable to tell us the pathway of this relationship, but that it exists there can be no doubt. The relationship has been but little heeded by physicians in general. Physicians are apt to tell patients to forget their ills when they are due to disturbances in the territory of supply of the central nervous system, but it is futile, because forgetting implies a mental process, and mental control over the sympathetic does not exist. We can, however, secure a certain amount of control over our wayward sympathetic nerve reflexes by getting under control our emotional state. The only truth in Christian Science lies in this, that the emotional state of the individual can sometimes by it be put at rest and so the reflex disturbances brought under control. Coué is quite right when he states that mere will power will not suffice to bring about a cure in this type of case. He regards imagination as a potent force, but, in reality, it is not imagination; it is equanimity or a complete emancipation from all emotional distresses.

In the lower orders of animal life the first development of the nervous system is really the sympathetic. Long before the

brain originates with conscious control of the body the excitative control of the unstriped muscle-fiber has been well developed. The annular ring about the esophagus in annelids represents with its backward running neurofibers the early stages of the sympathetic nervous system. In the gradual evolution of organic life the sympathetic nervous system is relegated to the background because of the development of the very much more important central nervous system with its control over the striped muscle-fibers and its function as the seat of the will. Because of the fact that the brain is so preponderant a portion of our nervous system, and because of the fact that our minds in general have so large a control over our bodies, we lose sight of the fact that another system exists within us which is just as important and which presides over the unstriped muscle-fibers of our body. The two systems seem to be quite distinct, and yet they meet on a common ground, and that is, that the mental state of the individual can react upon the emotional state of that individual. We cannot control our emotions, but we can put ourselves into such a condition that our thoughts exert a sedative effect upon the emotional state, just as we can put ourselves into such a condition as to permit our thoughts to exert a deleterious effect upon our emotional state. The ancient Hindoos with their ideas of perfect equanimity, persisting after death as the state of Nirvana, appreciated to some extent this relationship between the emotional state and the physical reaction.

If we are to treat mucous colitis, cardiospasms, spastic constriction of the colon, irritable bladders, and many cases of dysmenorrhea rationally, we shall be obliged to have in mind the relationship between these contractions and the emotional state. I do not mean to imply that there is no relationship between perversions of anatomy and reflex disturbances of the autonomic nervous system, but I do mean to assert that when disturbances of emotion take place, they themselves are frequently sufficient causes for the existence of the spastic states without the presence of any pathologic changes whatever.

Having had these considerations in mind in the treatment of

other cases of cardiospasms, we have been fairly successful. Some years since a patient who was sorely afflicted and who could not eat a meal without losing almost two-thirds of what he ingested came to me for relief, and was treated by the old method of cardiac dilatation. It worked like a charm for a few weeks, when gradually the old condition returned and cardiac dilatation had again to be resorted to. This was done three times in the course of some two years, when it became evident that the real advantage which accrued from this method of treatment lay in the fact that "it suggested to the patient that he could swallow." Thus, having faith that he could swallow, his emotional state of anxiety was brought under control, and consequently the reflex symptoms did not take place, and he swallowed without any trouble; but as time went on doubt began to appear as to the permanency of the cure, and with the doubt there came a recrudescence of the emotional anxiety, and with that, in turn, a reappearance of the cardiospasm. It became evident that to cure this individual another line of effort would be needed, and we therefore set about rather systematically trying to teach this individual the relationship which we knew to exist between his emotional state and his reaction. We got him to learn that if he could distract his mind by reading a book or a newspaper at the time he started to eat that he could at times go through a meal without regurgitating his food. We taught him when he was not disturbed by people looking at him, when he ate in private, when there was nothing to arouse his self-consciousness, that he could go through a meal with comparative comfort. By this method we induced him to gain several pounds in weight, and at the present time the man is living in comparatively good health, though he still finds it necessary to dine alone and to divert himself while eating.

It is very difficult at times entirely to eradicate from these individuals all emotional unrest and worry, and as long as any of it remains the symptoms may recur.

Another illustration of this relationship which exists between the emotional state and the symptoms is found in many instances of asthma. Physicians have long since noted the astonishingly

curious fact that asthma sometimes occurs at exactly certain hours at night. I have in mind at the present time a patient who as soon as the clock strikes 2 A. M. has or, rather, had his attack. He would go along reasonably well, dreading the appearance of the hour, and when the clock finally struck, knowing it was 2 A. M., the emotional anxiety reached such a high pitch of excitation that a reflex spasm of his bronchial muscles invariably followed. Many cases of spasms recurring at particular hours, at particular times, and on particular occasions all belong to this category of emotional reflexes and spastic contraction.

Feeling the truth of these facts, it seemed wise not to treat the patient whom we have just seen by the ordinary method of cardiac dilatation. It seemed wise rather to attempt to show the relationship which exists between the emotional state and the reflex cardiac symptoms. This was done, with very happy results. She gained very materially in her sense of well-being and in her weight. The look of her face became entirely changed. The apprehensions of death based upon the suggestive diagnosis of cancer of which she was aware gradually were given up as she kept living on and improving.

MUCOUS COLITIS

THE patient, a male forty-six years of age, weighs 155½ pounds. He complains of several bowel movements a day. All bowel movements contain mucus. He is extremely depressed. His weight used to be 190 pounds, but in the last few months it has been gradually falling, until now he regards himself as extremely thin. He has been at a hospital under treatment for four weeks, where he was put upon the so-called "colon diet" and where he was taught to be a "stool fan." His very eagerness to see the appearance of the stool led to the passage of several each day and to their very careful inspection on his part. Depending upon their appearance was his emotional state. If much mucus were present, he became extremely depressed. He lies awake all night thinking about his condition, and spends most of his day worrying about the mucus. He is a typical example of the fact that mucous colitis occurs in hysteric and neurasthenic individuals. Years ago when I first began the study of medicine Osler's text-book had just been published, and I remember the short account which this edition contained of mucous colitis. It was stated under the first heading, etiology, that mucous colitis was a disease peculiar to hysteric and neurasthenic individuals. Osler was certainly right. If I were to make the statement I should modify it by saying that mucous colitis occurs in individuals whose emotional state is disordered. In my experience mucous colitis never occurs except in individuals whose nervous organism is disordered, and usually it is disordered because of the disturbances which exist in the emotional state and the effect of this disturbance upon the unstriped muscle-fiber. In other words, mucous colitis is a pathologic expression on the part of the colon of a disordered emotional state. If the emotional state can be put into a condition of equanimity the mucous colitis will get well of its own accord. On the other hand, no amount of treatment of the colon, without putting at rest the emotional state of anxiety, will be of benefit. Colon

diets and bland diets are advisable, of course, but their effect is chiefly suggestive, and also there is the relationship between roughage—that is, food which has in it material which will irritate the lining of the bowel—and the reflex symptoms. It is right and proper in a condition of emotional unrest and worry that a bland diet be given, but reliance must not be placed entirely upon the bland diet, for until the emotional state is put at rest the mucous colitis will continue.

This patient was first seen at his home. His weight was 155½ pounds. It was very difficult to talk him out of his tendency to examine the stools and of his depression. I had almost to make fun of him by calling him a "stool fan" to get him to give up the idea of inspecting his stools. Within fourteen days he had gained 3 pounds and the bowel movements became less frequent in number. The depression was gradually growing less. He did not give way to tears as much as he did at first. He was given some extract of opium for the purpose of quieting his mental state of anxiety and not for the purpose of acting upon the bowels. By the end of three weeks his weight was 159 pounds and by the end of one month lacking three days his weight was 164 pounds. The patient stated that he was getting on well, that his insomnia was gone, that he was sleeping from six to seven hours, and that although he was still nervous he was gradually getting the best of his nervousness. At the end of the second month his weight was 168 pounds, the depression had largely ceased, and from being rather taciturn he had become slightly talkative. His stool was good and his bowels moved, as a rule, only twice a day. He was dismissed from the hospital with a weight of 168 pounds, and went to his home greatly encouraged and in a state of emotional calm.

Note.—This patient was an office patient for some time, and to substantiate the wisdom of this method of treatment is the fact that he now weighs 196 pounds and is entirely well.

CONCLUSION

These 2 cases which are shown are merely for the purpose of illustrating the general statement that the treatment of symp-

toms of unstriped muscle-fiber cannot be divorced from a consideration of the emotional state of the patient. If the patient is in a state of anxiety, worry, or grief, the condition in the territory of supply of the sympathetic nervous system cannot be very much obviated until the emotional state is put right. In our treatment of asthma, mucous colitis, spastic colitis, many cases of cardiac palpitation, many cases of irritable bladder, a good many cases of dysmenorrhea, and, indeed, even in some cases of spasms of the unstriped muscle-fibers of the gall-bladder, we must pay attention to the emotional state of the individual. If it is a young girl who is in love, if it is a man who is failing in business, if it is a woman who is distressed by her household cares or some domestic infelicity, we shall never succeed in getting this individual right in a physical sense until we succeed in getting him or her right emotionally. These cases are shown merely to make a plea that in general we pay more attention to the effect of the emotional state upon body functions; that we bear in mind that will power has no effect whatever upon the control of unstriped muscle-fiber; that unstriped muscle-fiber is really under the control of the emotions, and that, although anatomists have not yet pointed out the emotional center and do not know the pathways by which efferent impulses pass from this emotional center to the sympathetic nervous system, nevertheless such pathways exist and will some time be discovered.

CLINIC OF DR. WALTER W. HAMBURGER

MICHAEL REESE HOSPITAL

THE PREVENTION OF HEART DISEASE

No similar recent phrase in medicine has so caught the public's attention, challenged professional thought, or aroused such active discussion in medical circles as the slogan, "the prevention of heart disease." While distinguished physicians and public health officers state clearly their conception of this movement and relate the available measures which may be taken in the prevention of heart disease, equally distinguished members of the profession and medical editors support distinctly opposed views and refuse publication of material on this subject which they consider unscientific, inaccurate, and tending to mislead the public.

Such greatly divergent views on a subject which lends itself readily to scientific study and analysis are largely due to misunderstanding and misconception of the subject itself, the terms employed, and the aims of such an undertaking. It is my purpose to attempt to help clarify this subject from a clinical standpoint, to state my own views, to discuss in a practical way the procedures which are available at the present time in the prophylaxis of diseases of the heart.

At the outset I would emphasize that if something can be done from the standpoint of prevention this surely is a very much needed activity, inasmuch as progress in the control, treatment, and reduction in the mortality from heart disease in the light of present medical knowledge has almost ceased, so that in the attempt to reduce the mortality from this harvester of death the clinic must call upon the co-operation of social and public health agencies for assistance.

In outlining this subject for presentation and discussion I have arranged the material in the following outline which might serve in a general way as a program for such a campaign:

I. Reasons for a campaign for the prevention of heart disease; statistics regarding the morbidity and mortality of heart disease in the United States; prevention of disease in general; reference to various successful campaigns against disease; analogies and differences between the prevention of heart disease and prevention of other diseases.

II. Life-cycle of heart disease from its inception to complete decompensation; recoverability of heart disease; factors responsible for heart disease; popular misconceptions regarding heart disease.

III. Prevention of original involvement of the heart; infections—syphilis, thyroid disease, tonsils, rheumatism, typhoid, influenza; prevention of recurrences of heart involvement; predisposition of affected valves to new infections.

IV. Prevention of original heart muscle failure; prevention of recurrent heart muscle failure; infections, fatigue, toxic influences, poor hygiene; prevention of phobias and anxiety states in heart disease patients.

V. Paths and agencies through which propaganda concerning the prevention of heart disease should be pushed: physicians—education, training, medical societies; nurses, social workers, and their special societies; parents and parent-teachers' associations; schools—parent-teachers' associations, school survey, health examinations, etc.; industry—employees, periodic health examinations; special societies and cardiac clinics; co-operation with health organizations; press—popular and health magazines, public health lectures, etc.

I

Heart disease, according to Haven Emerson[1] in his Shattuck lecture of 1921, is at the present moment "the cause of the greatest number of deaths and disabilities in the United States."

[1] Prevention of Heart Disease—A New Practical Problem, Haven Emerson, Boston Med. and Surg. Jour., 1921, pp. 184–587.

Its pre-eminence as the "Captain of the Men of Death"—John Bunyan's phrase for consumption, Osler's for pneumonia—is due to a variety of causes, notably the decline in mortality of many other fatal diseases as a result of successful prophylactic measures and campaigns. In this regard we need only recall the brilliantly successful warfare waged against yellow fever, typhoid, malaria, hookworm, diphtheria, tuberculosis, to recount only a few of the more outstanding successes of preventive medicine. Statistics, as a rule, are tedious, uninteresting facts, yet in order that one may grasp the significance of this new practical public health problem a few facts must be given.

"About 5 per cent. of the men called in the drafts were rejected because of disabling heart disease." In the special cardiovascular examinations of approximately 10,000 troops of the Illinois National Guard in August, 1917, the special board which I had the privilege of directing accepted 2 per cent. conditionally for special light duty, rejecting 1 per cent. because of various cardiac disorders. About 2 per cent. of the children in school population are found to have some cardiac disorder. In the total population not more than 2 per cent. and not less than 1 per cent. is suffering from some form of organic heart disease, while one may say with reasonable accuracy that one in every 6 or 7 deaths is caused by cardiac disease. In other words, there are probably at the present time in the city of Chicago not more than 60,000, and not less than 30,000 cases of organic disease of the heart—while approximately 10,000 children of school age suffer with some form of disability referable to the heart.

There exists, then, at the present time a great public health problem, the extent and magnitude of which no one of us probably fully appreciates (accurate statistics are not available and are of great difficulty to obtain). In attempting its solution medical men are realizing the inadequacy of strictly medical preventive measures. Curative treatment is for the moment almost at a standstill. The clinic and laboratory at the present time have contributed all they possess to its prevention and control. We must go outside of the hospital and consulting room and enlist the community's help and co-operation in this pressing

need. This is peculiarly so in the case of the prevention of heart disease as distinguished from the prevention of many other disease processes, inasmuch as the causes of heart disease are multiple—the cause of many other diseases single. The control and prevention of heart disease, then, is the control and prevention of these multiple causes: rheumatism, St. Vitus' dance (chorea), tonsillitis, syphilis, the arterial changes of advancing years (arteriosclerosis), to mention only a few of the important etiologic agents.

II

Before we can discuss intelligently the prevention of heart disease we must have some knowledge and conception of the life-cycle of heart disease from its earliest inception (most often in childhood) to complete heart muscle failure (decompensation), often in middle life or early old age. This conception must include the commoner causes of the original heart involvement (in the young the major part is infective in origin), the frequent recoverability of the heart from these early infections—the frequent subsequent involvement and complications—the importance of school, social, industrial, occupational factors in these recurrences and complications, the frequent psychologic maladjustment in cardiac patients both young and old—and finally, certain popular and wide-spread misconception and traditions, current both among the laity and the profession, must be eliminated and made to disappear in the white light of modern sciences and investigation.

The word heart is said to be derived from a Sanskrit word meaning "to leap," probably from the jump or twist the heart gives at the time of going into contraction. By earlier peoples it was considered variously the seat of the soul, the mind, the spirit. Earliest Egyptian knowledge believed the heart was the seat of the soul. Greek civilization at the time of Hippocrates considered the head as the seat of the soul, the heart the domain of love and courage, the liver the organ of cowardice, jealousy, and anger. Our 20th century conception of the heart is that it is a muscular organ which is the propelling agent of the

blood in the animal body, a power chamber with the most delicate and efficient regulating mechanism working from before birth until the mortal part of us has no further need of it, capable of meeting the varied requirements of a varied existence, often in the presence of disease fighting against fearful odds.

Some of our modern misconceptions regarding the heart and its diseases are, first, that because the causes of heart disease are multiple (not single, as in tuberculosis, diphtheria, etc.), nothing, or little, can be done to prevent or ameliorate them. Nothing could be further from the truth, as I hope to show in due course of this discussion. Another frequent misbelief is that the heart has little or no recoverability from disease once it has become involved. In this regard there are certain definite organic involvements of the heart valves (subacute bacterial endocarditis), from which the organ may completely recover without any residuum or trace of the acute process. Other infectious involvement, such as acute, rheumatic fever, may leave permanent demonstrable scars on the valves, but for which the heart compensates and in spite of which the individual may carry on a long and fruitful existence as active as though the heart had never been involved.

These valve scars cause the popular "heart murmurs" with which the laity and the profession are so familiar, and which are so often so disastrously and mistakenly misdiagnosed and misinterpreted. These mistakes in interpretation and opinion are responsible for much of the physical invalidism and abnormal psychologic states which have ruined the lives and careers of many perfectly adequate individuals. One important angle of the prevention of heart disease, then, is the dissemination of accurate knowledge to the profession, so that such mistakes of diagnosis and opinion may be eradicated.

In the event that these valve scars are exceedingly widespread and multiple, particularly if the scars also invade the heart muscle, some degree of physical inadequacy may result (partial heart muscle failure, decompensation). Even here the patient is far from incapacited, it being only necessary that he carry on his activities at a somewhat lower level. Under careful

guidance and superdivision, adjusting his activities to his cardiac reserve, he may for many years carry on a happy and active, although somewhat diminished, career.

Other popular and enjoyable complaints which so-called heart patients indulge in are palpitation, angina, missed beats, fluttering, inability to lie on the left side, difficulty in breathing, sensation of the heart's beat, fear of sudden death. This discussion and elucidation would take too much time and are beyond the province of this discussion, only to say that for the most part they can be explained and understood on normal physiologic and psychologic mechanisms.

III

As has been intimated, the prevention of the initial involvement of the heart implies the prevention of the acute infectious diseases which leave heart residua, notably in childhood, acute rheumatic fever, chorea, tonsillitis, the common cold, diphtheria, typhoid, scarlet fever, pneumonia, influenza, etc. In early adult life certain streptococcus infections and syphilis, in middle life and old age the degenerative changes of arterial and kidney diseases.

Of the acute infections in childhood, rheumatism stands pre-eminent, and while influenza, diphtheria, and scarlet fever vary in their effects, rheumatism is constant. In an endemic of acute rheumatic fever in a children's hospital in London in 1919–20 Poynton found over half—66 per cent. of 172 cases had heart involvement, of which 22 children died and 38 were completely invalided. The rheumatic heart cases show a steady increase with school age ranging from two to twelve years, with many having the first attack at seven years, although it is relatively infrequent in children of school age of parents in better circumstances than the dispensary or hospital class.

The involvement of the heart from rheumatism, chorea, and tonsillitis is often extremely slow, insidious, and for a long time unrecognized. St. Lawrence, observing 65 cases of these so-called potential heart cases over a period of four and a half years, found 16, or 25 per cent., cardiac disease developed during

this time, and particularly in those cases where chorea was most evident. The preventive measures were most successful in the rheumatism, bone and joint pains (growing pains), sore throat, groups but of little or no value in chorea, while in this dread illness the valvular involvement known as mitral stenosis may not occur or be evident for months or years after the acute choreic process.

From the standpoint of prevention emphasis should be placed on the fact that rheumatism may be ushered in entirely silently, or with only a few "growing pains"—that the sensitive fidgety child may be in the midst of a grave chorea—that both rheumatic and choreic involvement of the heart should be recognized and treated at the earliest possible moment, instead of, as Wychoff has found, from four to eight years after its inception. Poynton has suggested that rheumatism be made a notifiable disease like diphtheria, scarlet fever, typhoid, etc., that schoolteachers be given simple lectures on rheumatism and chorea. Finally, care in the protection of other children in the household from the one with rheumatism and chorea may be an advanced, but not altogether, illogic precaution.

Considerable difference of opinion exists as to the value of tonsillectomy as a preventive measure. I confess to have seen severe initial and recurrent infections in tonsillectomized children with the same or greater frequency as in those in whom the tonsils have not been removed. Further tonsillectomy has, I believe, in a number of personal experiences been followed by an exacerbation of the original process.

Assuming that the original infection of the heart has come to a standstill, how frequent are recurrences and what measures are available for their prevention? Recurrences are frequent, and a valve damaged by an original rheumatic infection is more liable to a new involvement, particularly an involvement with a different type of infection (subacute bacterial endocarditis) than an undamaged valve. The precaution and measures for prevention of recurrences are in general the same as those of the original involvement, with perhaps even greater care and insight into the factor or factors responsible, including the careful

survey of hidden obscure foci of infection in teeth, tonsils, gall-bladder, prostate, thyroid, sinuses, etc.

IV

Aside from the attempts looking toward the prevention of the initial attack of heart pathology, as well as its recurrence, one of the if not the most vital function of the heart which it is our obligation to conserve and protect so far as possible, is the function of the heart which has to do with its action as a pump or power chamber, the propelling agent of the blood in the body. If this pump action remains unscathed in spite of initial and subsequent heart involvement all is well, if it becomes impaired (decompensation—heart failure) serious problems arise. The portion of the heart having to do with this pump action is the heart muscle or myocardium, and its impairment evidences itself clinically by such symptoms as breathlessness on exertion, easy fatigue and exhaustion, pallor or cyanosis, beginning dropsy of the feet and ankles, at times irregularity of the pulse and heart pain. The prevention of such heart muscle involvement resides, as in the involvement of the valves, in the prevention of the original and recurrent infections, but also notably in the adjustment of the patient's entire life routine, school, work, play, occupation, rest, recreation, housing, food, mental and physical activity, family, etc., etc., the myriad of social, domestic, economic, and industrial factors which affect the patient himself, his heart, and his circulation. It is in the prevention and direction of these factors that the social worker has been of such brilliant and effective usefulness and which as we gather data and experience in this field becomes of increasing value and necessity.

In the event that a state of heart muscle failure has occurred, such measures as partial or complete bed rest, hospitalization, digitalis, etc., become necessary for the return of the heart to compensation. In badly damaged hearts this bed rest may occupy weeks and months, and for such cases the need of prolonged convalescent care in hospitals or institutions for chronic cases, convalescent homes, and hospitals are urgently needed. Once such patients have recovered, they may return to their

activity if carried on at a somewhat lower level, but constant and often with careful supervision are liable to recurrence of their disability. These "repeaters" fill our hospital wards constantly, deprive the child of his schooling and play, the mother of the care of her home, and the father of his income from his occupation, and constitute the particular problem of the newly established cardiac clinic throughout the country. That such efforts are successful may be witnessed daily in these clinics, the efforts of one such clinic alone resulting in one year in the saving of upward of $25,000 pool income from the men thus enabled to continue at their work.

One of the really serious problems in all "cardiacs" and suspect cardiacs are the phobias and anxiety states which many of the patients develop or which the family and friends build up for them. The prevention of this abnormal psychologic situation, which often of themsleves cause partially a complete invalidism, lies largely in the experience and training of the physician, the nurse and social worker, their knowledge of heart disease *per se*, and their understanding, sympathy, and professional training in the hands of the patient himself. Untold damage has undoubtedly been done by ill-advised statements and advice, by grave countenances, and pessimistic prognoses, and, as one distinguished clinician has put it, he has "cured" more patients by telling them they had no heart disease than by any other method. Patience, insight, care and analysis of the individual, plus psychotherapy is usually all that is needed, although they require months and years for complete recovery.

V

The parties and agencies through which propaganda concerning the prevention of heart disease should be fostered are various. Perhaps first by the physicians themselves, through education, special training, medical societies, etc., nurses and social workers, parents and teachers, and by parent-teacher associations through schools, by health surveys and examination, through industry, arousing the interest of both employer and employee, by periodic heart examinations, special medical,

lay societies and organizations, by cardiac clinics, co-operation with various of the heart organizations, by the press, the popular and health magazines, public health lectures, etc. By many and all of these means much can be done from the standpoint of prevention, just how much no one can say as the attempt is just in its infancy and data difficult of securing and appraising. Not only are the results difficult of prophecy, but the extent and variety of the problem is surely not completely appreciated by any of us. It is to an interest and appreciation of this problem and a beginning attempt to attack it that this discussion is directed.

CLINIC OF DR. MILTON PORTIS

ST. LUKE'S HOSPITAL

MULTILOCULAR CYST OF THE PANCREAS

THE cases I am presenting to you today are from my private practice at St. Luke's Hospital.

This woman is seventy-one years old. She comes complaining of loss of weight and strength, anorexia, nausea, and weakness.

Present Illness.—She felt well until six months before entering the hospital, when she noticed that her appetite was failing. Soon after beginning a meal she had a feeling of fulness and weight in the upper abdomen. When she forced her eating she became nauseated. At no time, however, did she vomit. She has not had pain, but has had discomfort which she has associated with a feeling of gas pressure. She herself has observed that her weight was decreasing. She thinks that in the past six months she has lost 20 pounds. She has had chronic constipation for many years and this has been exaggerated in the past month. She complains of palpitation and of marked weakness. Two months after the onset of her present trouble she noticed a swelling in her abdomen and she was able to feel a mass that seemed to her to be about the size of an orange. She does not think that its size or shape has altered any since she first noticed it. At no time has it been tender. No special foods cause distress and none give her relief. She has not had any headaches and aside from a feeling of distention there has not been any pain at any time.

Past History.—She has had obstinate constipation for many years. She had pneumonia at the age of thirteen. For the past eighteen years she has had evidence of arthritis in the joints of the fingers and toes.

Family History.—Her father died of an accident at the age of sixty-one. Mother died at eighty of old age. She has 1 brother and 1 sister who are living and well.

Menstrual History.—Menses began at the age of twelve. They were regular and painless until she stopped menstruating twenty years ago. Since that time she has had no evidence of bloody or any type of discharge.

Physical Examination.—The patient is a poorly nourished white female and seems well preserved for her age. Her scalp and ears are negative. The eyes react in a normal manner, but she has a marked arcus senilis. There is no evidence of icterus of the sclerae. The tonsils are atrophic. The thyroid is normal in size. She has a slight enlargement of the cervical lymphatics. The lungs do not show any area of dulness. She has, however, scattered, coarse râles, more especially over the large bronchi. The heart is normal in outline and the tones are normal. The heart action is regular. Her blood-pressure measures 115 systolic and 68 diastolic.

The abdominal muscles are soft and flaccid. The subcutaneous fat is almost gone. There is a mass in the epigastrium which is about the size of an orange. It is not tender. The mass is movable, but seems fixed behind.

Rectal examination is negative. The reflexes are normal.

The fingers and toes show the typical deformity of chronic arthritis.

Urinalysis shows no albumin and no sugar, but large amount of indican and a trace of bile. Several hyaline casts were found in each microscopic field.

The stomach test shows a free acidity of 15, total acidity of 25. Occult blood was not present and pepsin and rennin were found in normal amounts. The duodenal contents show the presence of trypsin and amylase in normal amounts.

The stool examination does not show an excess of fat. Bile is present, but occult blood is absent.

The blood-count shows 4,700,000 red blood-cells, 7900 white blood-cells, and hemoglobin of 90 per cent.

The *x*-ray examination shows normal findings of the lungs.

The heart is normal in outline. The stomach shows no filling defect. The curve of the lesser curvature is widened by a mass which can be felt lying above and crowding into the stomach. In no position, vertical or horizontal, can a distinct filling defect be presented. The stomach empties in normal time. The bowel does not show any evidence of pathology. The opaque enema entered the colon without any obstruction. Both flexures are normal in contour and do not show any deformities.

The patient was referred to Dr. L. L. McArthur for operation. He found a strawberry-colored tumor, the size of a large orange, which took its origin from the tail of the pancreas. It contained many small cysts and the pancreatic tissue about the tumor was atrophic. The tumor was very vascular and was partially encapsulated. The pancreatic tissue could be pushed away easily except on the inferior surface, and in this place a resection was necessary. All exposed tissues and vessels were carefully ligated and the lesser omentum was closed tightly about the site of operation. A drainage-tube was inserted to the base of the site of operation.

The laboratory report of the tumor was as follows: "The tumor consists of an irregularly formed cystic tissue, measuring 7.5 x 6.5 x 4 cm. It weighed 87 grams. On the cut surface there is an edematous white tissue irregularly separated by septa. In some places there are some cystic spaces, 2 to 4 mm. in diameter. The microscopic report was that the tumor consists essentially of delicate hyaline fibrous tissue septa, some moderately thick, between which are cystic spaces from 1 to 3 mm. in diameter. These have a low cuboidal epithelial lining. A small mass of pancreatic tissue is attached to the tissue. A diagnosis of multilocular cyst of the pancreas was made of the tumor submitted."

Before operation the patient had a normal temperature, pulse, and respiration. The patient reacted unusually well from the operation and complained only of the usual abdominal postoperative symptoms. However, two days following the operation her temperature rose to 101.6° F. in the morning and in the evening it was 102° F. The patient complained of a cough

without expectoration. The respiratory rate was 24. Examination revealed an area of consolidation in the right lower lobe. Over this area she had dulness and bronchial breathing. Only the lower half of the right lower lobe was involved, but the following day the entire right lower lobe showed consolidation. Occasionally she expectorated a thick tenacious mucus which, on examination, showed many Gram-positive diplococci which resembled pneumococci. There was no evidence of tubercle bacilli. The temperature remained elevated for four days and gradually subsided, and at the same time the lung consolidation diminished and finally disappeared.

Unfortunately, on the seventh day after operation, the abdominal wound opened and there was a discharge of a fluid which was very irritating to the skin and caused some digestion of the wound along the opening. Some of the fluid was collected for examination and was found to contain both amylase and trypsin. The control of the fistula due to the pancreatic digestion was a very difficult matter. However, the drainage gradually diminished and the fistulous opening closed two weeks after its onset.

Multilocular cysts of the pancreas have been reported at various times, but resection of the pancreas for tumors has seldom been made. Finney in 1910 reported 16 cases and 1 of his own. Of these 17 cases, 9 recovered and 8 died following operation. The principal object of Finney was to report the successful removal of a primary solid tumor of the pancreas. The operation involved the resection of the entire middle portion of the gland, leaving only a small piece of the head and tail, requiring reunion of these by suture. Search of the literature reveals less than 20 cases in which the pancreas has been resected, either whole or in part, for primary tumor of the gland. Most of these were in women.

Finney's case was a woman of forty-three who came complaining of a "lump" in the stomach. She had had digestive disturbances for several years. She herself noticed a mass about the size of a small orange in the region of the stomach. It was not painful, but it was slightly tender and movable. She had

frequent and severe headaches, with nausea and copious vomiting. She lost weight and strength and had "sinking spells," which were frequent and severe. She felt better while at rest. Her bowels were obstinately constipated. Examination revealed a mass in the right hypochondrium, which was firm and nodular, about the size of a fist. It was movable and slightly tender. The urine was negative. Operation revealed the mass to be a tumor of the pancreas the size of a lemon, involving the middle portion of the pancreas. This was excised. The tumor had an indefinite capsule which could not be shelled out.

In his case, also, a fistula developed with copious discharge of pancreatic fluid for three months. The patient remained perfectly well after that time. Microscopic examination of the tumor proved it to be a benign cystadenoma of the pancreas.

As far back as 1884 Billroth removed the pancreas for adenocarcinoma and the patient recovered. In the Mayo Clinic in 1913 a case was reported in whom half the pancreas was removed for tumor. This case was very similar to the one which is here presented. It, however, was in a much younger woman, for her age was thirty-seven. She complained of severe attacks of pain which extended to the left side of the abdomen and the left lumbar region, and, besides, the tumor mass was very tender. The tumor was the size of a hen's egg. It was irregular and was located in the middle portion of the pancreas. The body and tail of the pancreas were removed. Microscopic examination revealed a benign growth which was thick walled, trabeculated, and contained numerous cysts buried in the serous pancreatic tissue.

Most of the cases reported in the literature proved to be carcinoma.

SURGICAL INDICATIONS IN PEPTIC ULCER

THIS man is forty-eight years old. He is complaining of epigastric distress, nausea, vomiting, dizziness, and fatigue. For the past twenty years he has had recurrent attacks of distress in his stomach, especially in the spring and fall. The discomfort is in the upper abdomen and it comes on one to two hours after meals. The taking of food brings relief and, likewise, the pain is relieved by soda. The pain he describes as a dull ache which at times becomes severe and it is accompanied by burning and gnawing. If he does not take food or soda to relieve his distress he becomes nauseated three or four hours after meals and vomits. Vomiting brings distinct relief. He has frequently been awakened in the early morning by the pain, which was relieved by taking milk or alkalies. He has lost several pounds in the past few months. He has gradually grown weaker and he has dizziness from time to time. His bowels are regular.

Past Illness.—He had measles and scarlet fever as a child and has had frequent sore throats. He had an initial lesion twenty-five years ago, for which he was treated for several years. He denies any other infections.

Family History.—His wife is living and well. She has not been pregnant at any time. There is no history of tuberculosis or carcinoma in the family.

Physical Examination.—The patient is moderately well nourished. Scalp and ears are negative. Pupils are small, but they react normally to light and accommodation. The reflexes are exaggerated. The thyroid is normal in size. The tonsils are moderately enlarged, but do not show any evidence of purulent material in the crypts. The lungs are normal. The heart shows a slight increase in the transverse diameter. The apex is in the mammillary line. The second aortic tone is distinctly accentuated. A systolic murmur is heard over the

base. The blood-pressure measures 130 systolic and 60 diastolic.

The abdominal contour is normal. There is no rigidity of the abdominal wall. There is distinct tenderness in the epigastrium and also some tenderness in the right lower quadrant. The liver is normal in size and it is not tender. The spleen cannot be palpated.

Rectal examination is negative. The stool shows a strong reaction for occult blood. The stomach shows a normal emptying time. Test of the fasting stomach contents shows a free acidity of 40, total 60. The Ewald test-meal shows a free acidity of 90 and total acidity of 130. It contains occult blood.

The *x*-ray examination shows normal findings of the lungs. The heart was dilated 10 per cent. in the transverse diameter. The stomach shows a distinct protrusion on the lesser curvature near its center. The diameter of the protrusion is $\frac{3}{4}$ inch, and it is typical of a penetrating ulcer. This region is very tender. The duodenal bulb is entirely normal in outline. The stomach empties in normal time.

Urinalysis shows a trace of albumin and a few hyaline casts.

The blood shows a red count of 3,700,000, leukocyte count of 9300, hemoglobin 60 per cent. The differential leukocyte count shows 73 per cent. polymorphonuclears, 25 per cent. small mononuclears, and 2 per cent. large mononuclears. The blood Wassermann test was clearly negative.

From the clinical findings and laboratory and *x*-ray evidence it is distinctly evident that the patient has a penetrating gastric ulcer. Because of the large size of the ulcer and the age of the patient the question of malignancy demands serious thought. I advised the patient to have an operation instead of going through the usual medical course of ulcer management.

He was operated on by Dr. L. L. McArthur, and a large penetrating ulcer was found on the lesser curvature just in the place indicated by the *x*-ray. The ulcer was resected and the site was sutured. The laboratory report is as follows:

"This is an ovoid tissue 2 x 2.3 cm. and 1 cm. in thickness. On the inside is an ulcer 1.5 x 1.5 cm. and depressed in the center

for about 0.5 cm. There is considerable scar tissue between the base of the ulcer and the under side of the tissue. In the base there is an opening of a vessel with a pin-point size lumen containing a blood-clot.

"*Histology.*—In twenty sections taken at different levels from different blocks of the ulcer crater, grossly there is chronic inflammation and there are regions of chronic granulation tissue well healed and extending deeply into the muscularis. There are no changes in the sections suggestive of carcinoma."

The patient is making a splendid recovery from the operation. He will observe a careful diet with alkaline therapy for a number of months to prevent recurrence of the ulcer or the formation of one at another site.

The question of medical or surgical treatment of peptic ulcer has been discussed a great deal in recent years. The indications for medical and surgical treatment of peptic ulcer vary decidedly when they are stated either by the internist or by the surgeon. There is no such thing as exclusive medical treatment and, likewise, an ulcer is not always a surgical disease. The recommendation in a given case depends upon the size, type, and site of the ulcer. All surgeons agree that acute, uncomplicated ulcers are best treated medically. It is only in the long-standing chronic ulcers, especially in those with complications, that surgical intervention is indicated. Most medical men agree that surgery should be resorted to in all ulcers that have failed to heal upon a thoroughly applied course of medical treatment given for a sufficiently long time. Likewise, an exploration is demanded in all cases in which there is a suspicion of malignancy. Operation should also be advised in all cases of repeated menacing hemorrhages which have recurred in spite of careful medical management. Acute perforation is always a surgical complication, and in chronic ulcers with definite manifestations of subacute perforation surgery usually offers the best result.

High-grade pyloric obstruction, hour-glass contractions of the stomach, and disabling perigastric adhesions which interfere with proper drainage of the stomach should all be treated surgically. Cases with coincident lesions in the gall-bladder,

appendix, pancreas, or pelvic organs are best taken care of by the surgeon. When surgery is resorted to our best results have been in the cases in which radical resection of the ulcer was carried out. Pylorectomy, as done by Polyá, and pyloroplasty, based on the Finney or Horsley type of operation, are distinctly more physiologic in their action than gastro-enterostomy. When gastro-enterostomy has been done we have had the best results in those cases in which the pylorus was closed off at the same time. We do not know the exact cause of peptic ulcers, and until we do learn their exact etiology our treatment from both the medical and surgical standpoint will be a prolonged slow affair. Because of the evidence in favor of infection as an important etiologic factor, we must be careful to have all focal infections, especially those of the teeth and tonsils, eliminated before a medical course of treatment is begun. In cases of long-standing gastric ulcer, which fortunately in our experience is much less common than duodenal ulcer, we are less inclined to carry out prolonged medical treatment because of its tendency to undergo malignant change. Even though a number of observers have demonstrated with the *x*-ray that the large craters of such ulcers can be healed by medical treatment, we still feel that it is impossible to differentiate by the *x*-ray benign and malignant ulcer, and to give the patient the benefit of the doubt we resort to surgery at once. In cases of severe hemorrhage medical measures offer the best result, and it is only when repeated hemorrhages occur that surgery is indicated. Moynihan, in the Lancet of last year said, "I think it a reproach to medicine that surgeons should be compelled to operate so frequently on gastric and duodenal ulcers when such an ulcer should be cured far more often than it is by medical treatment. Physicians who acquaint themselves with the living pathology of gastric and duodenal ulcers realize how protracted the medical treatment of these complicated lesions must necessarily be." This, it seems to me, is a fair statement of the case. We must learn to diagnose our ulcers at the early stage and institute thorough and persistent medical measures at once and thus prevent the later complications. Until we can do so the medical men must admit their failure in a

percentage of ulcer cases and call upon the surgeon to help. Unfortunately, the majority of patients with ulcer are not given a rigid and systematic course of treatment, and it is not fair for the surgeon to say that ulcers cannot be healed by medical management when his observations are based on patients who have not had the benefit of intelligent medical measures.

CLINIC OF DR. CLIFFORD G. GRULEE

PRESBYTERIAN HOSPITAL

ACUTE HEMORRHAGIC NEPHRITIS IN CHILDREN

THIS little girl, Louise G., two years old, entered the hospital on February 11th this year. The history is very accurate, because the mother is a trained nurse and has been very observing. It is as follows: About two weeks before the child entered the hospital the mother noticed that it had marked enlargement of the cervical glands and that "they stood out in little bunches" on both sides of the neck. Since the child did not complain about them, no further thought was given to them. About two days later the urine suddenly became bloody and very scant, the child voiding only between 3 and 4 ounces a day. It continued about the same for five days, and on examination showed albumin, blood, casts, and pus. The blood gradually decreased until the urine regained its normal color, but it remained cloudy and scanty in amount.

Then, without any complaint of pain on the child's part, the ear began to discharge. Immediately the blood returned in the urine, and at this time she was brought to the hospital. Previous to this illness the child has always voided a large amount, especially at night. There is no pain associated with the passing of urine, but there seems to be some difficulty, and it requires considerable time for the child to void even a small amount. During the past two weeks the appetite has been very poor. She has vomited occasionally after eating and has been quite constipated, the stools being clay-like, having a foul odor, and containing mucus and curds.

For the past two months the mother thinks the child has been losing weight. One month ago she weighed 25¼ pounds,

and on entrance she weighed 23 pounds, 1 ounce. Cough has been persistent, being accentuated by the child's tendency to catch cold.

Past History.—A normal baby, full term, transverse presentation. This is the second child. Breast fed for a month, and after supplementing for three months was then put on formula. At nine months the child had chickenpox, and at one year had a condition which was diagnosed by competent observers as smallpox. Measles at fifteen months; continuous colds all winter.

Family history shows nothing of any significance in this case.

In summing up the history, then, we have a child two years old which, during the entire winter, has been subject to colds. During the past month there has been a rapid loss in weight and evidently considerable cough. Two weeks before entrance the mother noticed enlarged cervical glands, and two days after this a bloody urine. This urine was shown to contain albumin, blood, casts, and pus. The bloody color of the urine disappeared, to suddenly reappear at the time of the occurrence and rupture of an otitis media. The urine was bloody and scanty and passed with some difficulty, but with no pain.

Now, when we look at this child with a history of so much illness and with the record of a bloody urine for almost two weeks, we are struck with the fact that the child shows practically no prostration. She is naturally very subdued in manner and not demonstrative, so that the calmness with which she takes her new environment is not the result of any prostration, but is simply a matter of temperament. It is noted that there is present a certain amount of pallor. This pallor has not the yellowish tinge which we are accustomed to speak of as cachexia and which is frequently encountered in severe anemias, but is a pallor which is often found in childhood and whose significance cannot be judged until a blood-count is made. Such a pallor as this child shows is very often found in children with a perfectly normal blood-count. We can, therefore, see that this pallor may either represent a moderate degree of anemia or it may be due to internal congestion with consequent contraction of the

peripheral blood-vessels. When we examine the head and neck we find the tonsils are large and red and the cervical glands are readily palpable. There is also a discharge from the left ear. The examination of the thorax shows nothing in the lungs. Over the heart area there is a slight systolic murmur, and an irregularity which corresponds in time with the respiration. In other words, a sinus arrhythmia. In view of the fact that the heart is not enlarged and that the pulmonic second tone is not accentuated, it seems fair in this case to regard the murmur as of the adventitial, or so-called hemic, type.

The abdomen shows the liver palpable somewhat below the costal margin. The spleen is not enlarged; the kidneys are not palpable and there is no tenderness over the kidney region.

Another point I wish to call your attention to is the fact that there is no palpebral edema, nor is there any edema of the ankles. In other words, there is not now, nor has there ever been, any edema in this case.

We find the following notations of what has occurred since entrance:

On February 12th the urine was still scanty and bloody; the child looked quite pale.

On the 16th the urine has improved steadily. Excursions of temperature, which I will come back to in a minute, are still quite marked General condition seems little improved.

On the 19th we find the note that the child is generally improved. She is conversing with other children in the ward more frequently. The leukocytes have decreased in the urine and blood is only present in microscopic amounts.

On the 21st the child is not so well. She vomited twice; the left ear-drum was slightly reddened.

On the 27th the temperature arose abruptly; the left ear-drum was punctured by Dr. McGinnis; the temperature dropped, but none of these conditions had any apparent effect upon the urine.

On the day of entrance the blood showed 4,260,000 red cells, 13,200 white cells, and 85 per cent. hemoglobin. The tabulation shown here (indicating blackboard) will give you perhaps a

better idea of the changes in the urine than would reading out those changes from day to day.

TABLE

Date.	Nucleo-albumin.	Serum albumin.	Benzidin test.	Casts.	Number of leukocytes.	Microscopic blood.
2/12	5 mm.	?	++++	+	820	++++
2/13	8 mm.	Trace.	++++	0	530	++++
2/14	3 mm.	Trace.	+++	+	480	+
2/16	Trace.	0	+	Few.	110	+
2/17	0	0	?	0	60	Few.
2/18	Trace.	0	0	Few.	70	0
2/19	0	0	0	0	50	0
2/20	0	0	0	0	20	0
2/22	0	Trace.	0	0	40	Few.
2/27	0	0	0	0	50	Occa-sional.
3/2	0	0	0	0	50	0
3/6	Trace.	Trace.	0	Occa-sional.	40	0

It is extremely difficult in these small children to estimate the quantity of urine passed, and this, of course, is very necessary in cases of this type. It can only be said in a general way that by the 17th the urine was passed much more frequently and in larger quantities, and that it was noted that while in the early specimens the specific gravity was as high as 1.026, on this date it dropped to 1.008. Subsequent to that date the quantity of urine obtained for examination was too small to estimate the specific gravity by ordinary laboratory means.

I wish now to call your attention to the temperture curve in this case. The condition which we have in mind is quite characteristically represented by very marked changes in temperature, and these are very beautifully shown in the first week of the illness of this child while in the hospital. You will notice by this chart (Fig. 127) that the variation in the extremes is from 96° to 102° F., but more characteristic than this variation is the sudden rise and fall in temperature. This frequently amounted to 3 degrees in a very short time. Naturally, the

acute attacks of otitis media were accompanied by high rises in temperature, but such rises do not show the same irregularity in the temperature curve (Fig. 128), the temperature being maintained until the ear began to discharge following paracentesis.

It is very evident that we have here a case of acute hemorrhagic nephritis in a little girl two years old. The very definite

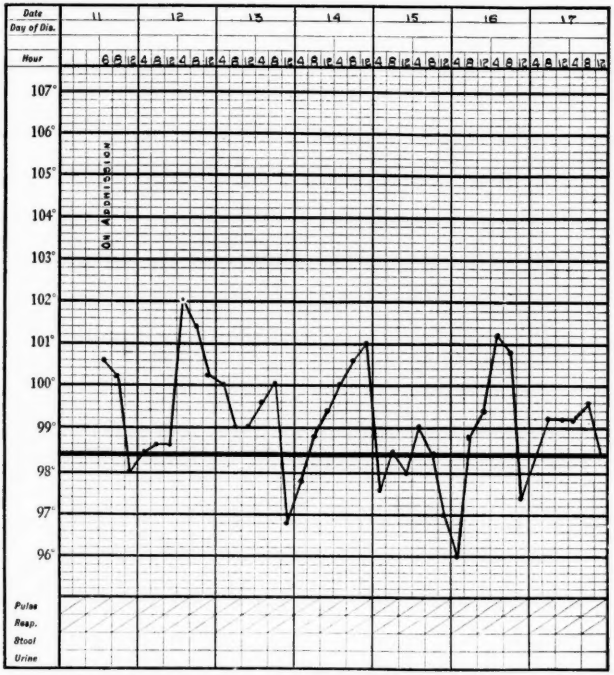


Fig. 127.—Temperature curve during acute stage of disease.

connection of this condition with infection of the throat, and secondarily in the glands of the neck at the time of first appearance of blood in the urine, and on the second occurrence of that blood, the development of an acute otitis media, point at once to these infectious conditions as the cause of the disorder.

In 1915 Dr. Gaarde and myself reported several cases of

this condition in which there was quite regularly found the same organism in the throat, or other focus of infection, in the urine and in the blood. In order, however, to obtain positive cultures from the blood and urine it is necessary that these be examined within twenty-four to forty-eight hours after the appearance of the bloody urine Cultures obtained later show no growth.

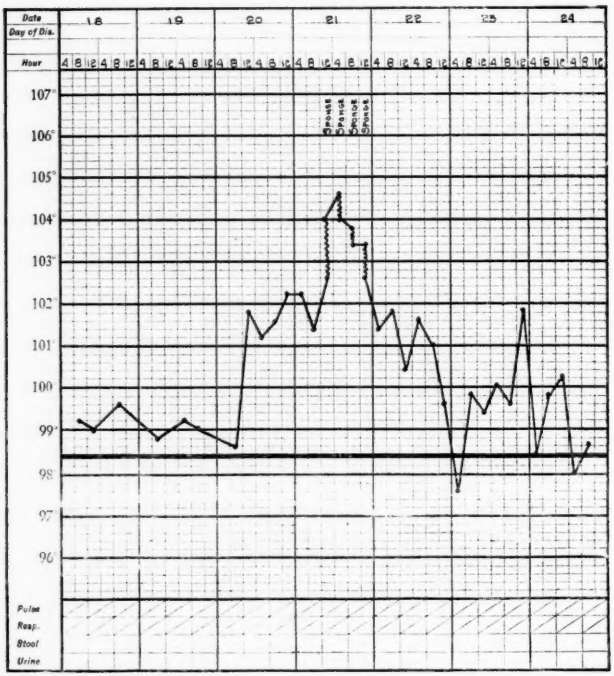


Fig. 128.—Showing a rise in temperature as the result of otitis media.

It was, therefore, very evidently impossible in this instance to get results by such means, and they were not attempted.

The course in this case is peculiar, in that it is interrupted. Usually in these children the blood quite rapidly disappears from the urine and is replaced by an increase of the leukocytes, as in this instance. The peculiarity in this case lies in the fact

that a secondary infection produced a recrudescence of the kidney condition. I have never seen this occur before.

This represents the most common form of acute nephritis which we encounter in childhood. It is seen usually between the ages of two and six, though I have seen cases in children under six months. There is no special predisposition so far as sex is concerned, but, as might be expected, we encounter it more often during the cold months of the year when infections of the upper respiratory tract are so much more frequent. In the absolutely typical case where a very definite history can be obtained it is usually found that there has been an acute tonsillitis or otitis media from ten days to two weeks previous to the appearance of the blood in the urine. As in this case, the glands of the neck are frequently involved, or there may be an involvement of the mastoid process.

The temperature following these acute conditions may or may not have subsided, but with the appearance of the bloody urine there is always a rise in temperature, and the temperature curve takes on the general characteristics of that in the case before us.

The onset of the condition is not accompanied by any special prostration on the part of the child, and is usually first noticed by the attendants only because of the appearance of the bloody urine. One can scarcely make a mistake in this. The urine contains large quantities of blood, is dark red, and as the blood decreases becomes smoky and then clears up. The general course of the condition is such as we have seen in this instance except for the interruption which was caused by the appearance of the otitis media. That is, the temperature subsides gradually within a week to two weeks, as a rule, during which time the blood slowly disappears from the urine, and as the blood-cells decrease the leukocytes increase. The albumin and casts rather rapidly disappear, the quantity of urine, which at first is scanty, increases and, finally, the number of white cells decreases and the child is discharged with a urine which, so far as can be determined, is normal. Only in rare instances where the course is quite prolonged do we get evidence of a chronic infection of

the kidney. This, in my experience, has certainly not occurred in 10 per cent. of the cases. In this instance, while the cellular elements disappeared quite rapidly, there remains some albumin and an occasional cast and the quantity of urine passed is rather reduced.

One is struck by certain features of this clinical condition. The appearance of the bloody urine is nearly always a cause for alarm, and one is very much surprised at the absence of physical signs and symptoms which are associated with nephritis in adults. There is apparently no tendency whatever to the occurrence of uremia, nor are there any evidences of edema.

No blood chemistry was carried out on this case because of the difficulty of obtaining blood from a child of this age, and also because there were never any clinical signs which would lead one to think of a severe impairment of the kidney function as manifested by its effect upon the general economy of the child.

The **diagnosis** of this condition is not difficult. The child is practically always brought to you with the diagnosis already made, in that the mother always speaks of the occurrence of the bloody urine. No other condition with which I am familiar will produce in these children such large quantities of blood without any apparent cause. Vesical calculus is rarely if ever the cause of such a hemorrhagic urine and is nearly always accompanied by rather severe pain. Vesical polyps are extremely rare in children, but it is conceivable that this condition might produce a clinical picture with which acute hemorrhagic nephritis would be confused. With these exceptions I know of no other condition which need even be considered at this age.

The **prognosis** is such as I have already pictured to you. Nearly all of these cases recover without impairment of kidney function. There is, however, a small percentage where there seems to be permanent damage to the renal tissue, so one must be somewhat guarded in giving an absolutely good outlook.

Treatment must be considered under two heads—the medical and surgical. The medical treatment consists of such measures as will alleviate the kidney condition. The surgical treatment

is directed toward the removal of the focus of infection. There must be some question as to whether we should immediately remove the focus of infection in these cases. It has been my custom to counsel more conservative measures. In other words, to treat the nephritis medically until it has cleared up, and then remove the focus of infection. That something, however, may be said in favor of the other course is shown by the fact that in one case which came under my observation where an acute mastoiditis developed with the appearance of the bloody urine and demanded immediate operative interference, the urine cleared up very rapidly following the operation, nor did there seem to be any apparent damage done to the kidney condition by the anesthetic or operative procedure. It would seem, however, in the majority of cases that it would be better to treat them as a nephritis first and remove the focus of infection later, where possible.

The medicinal treatment is very simple The most that one can do is to give these children small quantities of alkalies. I usually use sodium bicarbonate or potassium citrate in sufficient dosage to keep the urine slightly alkaline. Too much alkali should not be given, and the attendant should be instructed to test the urine whenever it is passed. I would strongly advise that these alkalies be given every four hours, night and day, so that the urine may be continuously alkaline, and not be alkaline part of the time and acid the rest. Water should be given in rather large quantities and the diet should be plain, consisting altogether of milk and cereals in the acute stages.

For the temperature lukewarm sponges and alcohol rubs are usually sufficient. The child should be kept carefully in bed and should not be allowed to move about. Chilling should be avoided in every way and, of course, cleanliness of the skin is absolutely essential. After the disappearance of the blood and when the leukocyte count is reduced, if the temperature has reached and remained normal for several days, then one should consider the treatment of the focus of infection. This, in the majority of the cases, is the tonsil. Of course, where the infection is situated elsewhere there may be some difficulty in eradicating it entirely.

CLINIC OF DR. JAMES G. CARR

COOK COUNTY HOSPITAL

SECONDARY HYPERTROPHIC OSTEO-ARTHROPATHY

THIS boy whom we present to you this morning is thirteen years of age and, according to his own statement, has had "tuberculosis" for five years. He has been in several "fresh-air hospitals." He attributes the onset of his illness to an attack of influenza which he suffered some five years ago, since which time he has never been free of a cough, worse at night and early in the morning, productive of a thick, greenish sputum, in quantity often amounting to half a cup a day. Sometimes the sputum is bloody; about two years ago he had one frank hemoptysis. In addition to the cough he has had shortness of breath upon exertion, pain in the right side of the abdomen, and swelling and pain in the extremities, especially at the ends of the fingers. He has also had night-sweats, sometimes drenching in character, throughout the course of his illness. The swelling of the fingertips and the extremities began early in the course of his disease. The joints are tender and sore upon pressure and motion. Recently the extremities have been worse, the pain and tenderness being aggravated, and the enlargement increased. During the past month he has also had a diarrhea. His appetite has been poor recently, and with the aggravation of his other symptoms his cough has been worse and he has often vomited after severe coughing spells. He sleeps well, save for the fact that he is often obliged to get up at night to urinate.

Except for the influenza, to which reference has already been made, his history is negative, and there is no family history of tuberculosis or of any chronic pulmonary disease.

Status Præsens.—A poorly nourished and poorly developed boy, much under size for his age. The teeth are not in good

condition; the tonsils are present, fairly large and full of crypts. The cervical glands are enlarged. The lungs are hyperresonant throughout; the chest is barrel shaped. The breath sounds are harsh, especially on the right side, and the expiratory murmur is prolonged. Numerous medium moist and coarse dry râles are heard over both sides, but particularly over the right.

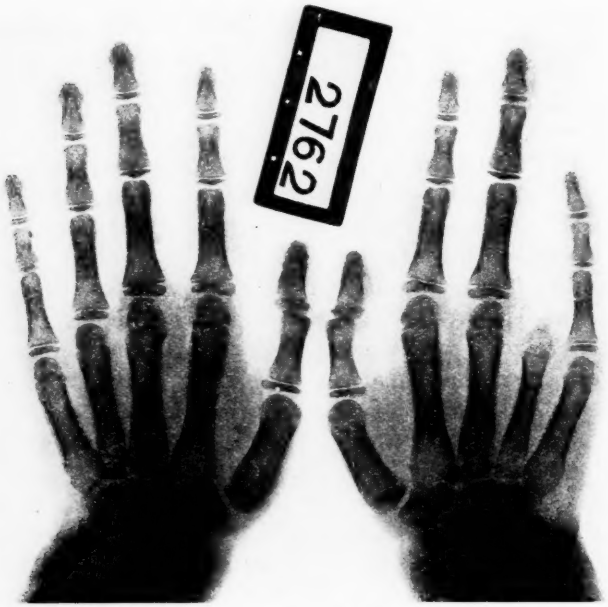


Fig. 129.—Radiograph of hands: Note wide distal phalanges and periosteal proliferations of metacarpal bones.

The excursion is limited over both sides. Except for an increased rate, the cardiac examination is negative. There is general abdominal tenderness, without any localization of the same and without rigidity. There is thickening at the lower ends of the humeri and ulnæ, with associated swelling of the joints, which are tender to pressure and painful upon motion. There is marked clubbing of the terminal phalanges of the fingers of both hands.

There is tenderness and swelling of both hips, but without disturbance of function. The ankles and knees, as you can all see, are swollen, and you can likewise note the tenderness to pressure. The legs are swollen from the knees to the ankles; there is an edema which readily pits upon pressure. The pressure causes pain. Both feet are involved in a manner like that of the hands.

The urine is negative. The blood-count shows: Hb. 60 per cent., erythrocytes 3,190,000, leukocytes 4500. The blood Wassermann is negative. The blood-pressure is 100/60. Three examinations of the sputum have been made; in none was the tubercle bacillus found.

Let us omit for the present a discussion of the nature of the pulmonary lesion which may be regarded as the primary disease, and turn our attention to the disease of the extremities with which this boy is afflicted. The changes in the extremities are obvious, the wide terminal phalanges of the hands and feet, typical "clubbing," with enlargement at the joints of the hands and feet, the knees, ankles, wrists, and elbows. We have here the *x*-ray pictures of the hands. The report reads, "There is noted clubbing of the soft tissues of the terminal phalanges of the hands, with accompanying curvature of nails. Shafts of radii and ulnæ in small portions included upon the plate appear stocky and with shafts of all of metacarpals show periosteal proliferation, the whole constituting the picture of pulmonary osteo-arthropathy."

In March, 1889 Bamberger reported to the medical society of Vienna 2 cases of bronchiectasis with wide-spread involvement at the joints. In both of these the ankles, wrists, and elbows were affected, but in one there was also tenderness over the thighs and patellæ. One of the patients was a man of twenty-five, and the pulmonary disease had lasted eight years; the other was a man of forty-eight, who had been sick one year. Bamberger stated that he had seen 2 similar cases the year before, and that one of these had come to autopsy. The bones of the leg were found to be thickened. One striking observation was made, the full purport of which will be better appreciated with further consideration of the subject. "These cases may be re-

garded as a diffuse hypertrophy of the bones, the etiology of which, in most cases, is uncertain." Bamberger noted the occurrence of this condition with "clubbing," and was the first to raise the question as to the pathogenesis of the changes, the same question which will engage our attention today, as to whether or not the disease is the result of a toxin or of passive congestion. It is curious that while the clubbing of the fingers had been noted by the ancients, the associated changes in the bones of the extremities had not been noticed prior to Bamberger's report. In the following year Marie published a comprehensive report of 8 cases, describing in detail the changes in the long bones. Since the appearance of his paper the condition has often been called "Marie's sign group." Though all of Marie's cases will not stand the test of rigid criticism (at least one of his cases is now believed to have been a case of acromegaly), he is credited with the first comprehensive description of the disease, and the name which he proposed, "osteo-arthropathie hypertrophiante pneumique," is the most generally used designation for the condition. Translated into our own language, Marie's disease is known as "hypertrophic pulmonary osteo-arthropathy." Marie emphasized the relationship of the disease to primary pulmonary disease. Since Marie's publication there has accumulated a large volume of literature much of which is purely speculative. The clinical and pathologic features of the condition are well established. It is in connection with the etiology and pathogenesis that dispute has been most constant. Another subject of discussion has been the relationship of "clubbing" of the fingers to Marie's disease. It is now the generally accepted opinion that these two conditions are but different stages of the same disease. Sternberg classified the disease in three groups: (1) pure clubbed fingers, (2) clubbed fingers with transitory periosteal deposits on the distal ends of the bones of the extremities (Bamberger's type), (3) finally, the severe picture, in addition to clubbed fingers, high-grade changes in most parts of the bony system (Marie's type). Högler quotes this classification of Sternberg, and adds that a fourth type must be admitted to cover a type of case reported by

Schlagenhaufer, in which there were typical periosteal deposits without clubbing. In this connection it is interesting to note that Locke quotes Landis, to the effect that "clubbing of the fingers invariably occurs in secondary hypertrophic osteo-arthropathy."

Hypertrophic pulmonary osteo-arthropathy may be defined as a disease of secondary nature occurring in the course of various chronic conditions, characterized clinically by enlargement of the extremities, with clubbing of the fingers and thickening at the joints or about them, and anatomically by thickening of the soft parts, especially of the distal phalanges and proliferative processes of the periosteum, particularly at the lower ends of the bones.

The pathologic changes which this condition presents may be broadly divided into two groups: (1) those with clubbed fingers only, (2) those in which the bony changes are well marked, along with the clubbing of the terminal phalanges. In a very considerable proportion of the cases changes in the soft parts are alone responsible for the clubbing of the fingers. Vascular dilatation is chiefly responsible for the increase in size, though there is probably some connective tissue increase in many cases. The changes in the nails, enlargement, and increased convexity are characteristic and occur early. The recognition of the ungual peculiarities is attributed by Ebstein to Hippocrates, who noted its association with chronic empyema. Bony changes in the terminal phalanges do occur, though the frequency of such changes is still a matter of dispute. Bamberger found such changes in one of the cases mentioned in his first report. This proliferation of the periosteum of the terminal phalanges is more likely to be found in association with wide-spread osseous pathology in such cases as Sternberg included in his third group. Locke believes that careful *x*-ray study will reveal alterations of the long bones in many of the cases which clinically show clubbing only; these cases are covered by Sternberg's first and second groups. On the basis of radiologic studies, Kessel has shown that "bone changes occur more commonly than is ordinarily supposed in the so-called simple club fingers." That the

characteristic changes in the long bones may be demonstrated by the *x*-ray in cases which show clubbing, but without the clinical signs of alterations in the long bones, has been shown both by Locke and Kessel.

The osseous changes occur most frequently and characteristically in the bones of the extremities. Of these, the bones of the hands and feet, phalanges, metacarpals, and metatarsals, the lower ends of the tibia and fibula, the radius and ulna are most likely to be affected. The femur and humerus may be involved, and in cases of extensive disease the bony alterations may be found in almost all the bones of the body. This latter is very rare. The essential bony change is a proliferation of the periosteum. The thickening of the ends of the bones, especially of the lower ends, is especially characteristic of hypertrophic pulmonary osteo-arthropathy, though the proliferations are not confined to these localities. Irregular thickenings, due to this periosteal proliferation, may be found through the length of the bone. Kauffmann makes this statement: "At times, throughout their entire length, these bones (of the leg and arm) may be covered with flat periosteal deposits." Many writers have used the term "ossifying periostitis" to describe the typical periosteal pathology. That is the term used by Ebstein in his comprehensive review, and Locke uses the same words to describe the "typical process" in the bones. Högler objects to the term "periostitis" as signifying an inflammatory process. He denies that such is present. To quote him, "Roentgen examination shows that the shaft of the bone is intact and that the thickening rests exclusively on the periosteal deposits described. These deposits are hyperplastic, not inflammatory."

As to the share of the joints in the pathologic process, there is also much dispute. Kauffmann says, "Spindle-shaped enlargements of the bones making up the joints produce the essential joint changes which are observed in severe cases, while only in rare cases is the cartilage eroded," and adds parenthetically, "Thus the term 'osteo-arthropathy' is too comprehensive and the suffix 'pulmonary' is too restrictive." On the other hand, Locke, in the most complete and modern discussion of the sub-

ject which I have seen, says, "Recent authors nearly all agree that at least in all well-developed cases the joints themselves participate in the morbid process." So far as our patient is concerned, the signs of a true arthritis are obvious to you all. The elbows and wrists, the knees, and ankles are swollen; they look and feel like joints distended with fluid.

The whole tendency of the studies directed toward this subject since the time of Bamberger has been to identify as one disease clubbing of the fingers and the bony changes of the extremities described by Bamberger and Marie. Bamberger taught that the so-called club fingers, even when they are the only demonstrable change, are to be regarded as the beginning stage of Marie's osteo-arthropathy. It must not be forgotten that separate descriptions of the two conditions or classifications such as that of Sternberg (which is useful for a presentation of the subject) may be misleading if they serve to cause any confusion in your minds as to the essential unity of these conditions. When we come to consider the pathogenesis of the disease, we will be confronted again with the temptation, in order to account for the effects of two widely different causes, to accept the duality of the two conditions. We want to make it plain, perfectly plain, that in the minds of the most careful observers hypertrophic pulmonary osteo-arthropathy is one disease which presents two stages; often the first stage exists long before the signs of the more advanced stage are present.

In the consideration of the etiology it must be emphasized that hypertrophic pulmonary osteo-arthropathy is a secondary disease. There is thus far no proof that the condition is ever primary. Locke has analyzed 21 cases in the literature "reported as examples of secondary hypertrophic osteo-arthropathy without relation to any antecedent disease," and found that in only 5 of these was there no history of a primary disease, and in none of the 5 was there a postmortem record. We quote his conclusions: "We know that in the overwhelming majority of cases Marie's disease is characteristically a secondary condition, and are justified, therefore, in demanding complete proof that any given example is primary. The 5 cases above mentioned,

while seemingly not secondary to any other disease, can be regarded as only suggestive of the possibility of a primary form of hypertrophic osteo-arthropathy. Until positive proof is forthcoming that such a primary type does exist we may assume that the condition is always secondary."

The conditions to which hypertrophic osteo-arthropathy is secondary are many, but certain chronic diseases of the heart and lungs are most important. Chief among these are bronchiectasis, pulmonary tuberculosis, chronic empyema, and congenital heart disease. Numerous intoxications and infections have been cited from time to time as causative factors in the production of typical clubbing and bone changes; among these are chronic jaundice, chronic alcoholism, pyloric obstruction, enterogenous cyanosis, pyelonephritis, dysentery, syphilis, purpura, malaria, and polycythemia, with enlargement of the spleen. It is interesting, in reading over the literature, to note the changing views in regard to certain phases of the etiology and at the same time to recognize how continually certain diseases are admitted as the outstanding causes of the condition. Thus, in 1897, Teleky published the following classification: (1) Diseases characterized by purulent and gangrenous processes in the body, pulmonary tuberculosis (with cavity formation), bronchiectasis, empyema, cystopyelonephritis, dysentery; (2) infectious diseases and chronic intoxications: pneumonia, pleurisy, syphilis, and alcoholism; (3) cardiac diseases, especially congenital; (4) malignant tumors, pulmonary sarcoma, pulmonary carcinoma, sarcoma of the parotid; (5) diseases of the nervous system; syringomyelia, neuritis.

Locke accepts this classification as "the most satisfactory grouping of the causative diseases," but ignores the fifth group (concerning nervous diseases); this is in keeping with present-day opinion that nervous diseases do not cause osteo-arthropathy. Högler, concerning whose opinions we will have more to say later, proposes a classification which omits cardiac disease as an essential group: (1) Chronic suppurative pulmonary disease, (2) malignant tumors, (3) biliary cirrhosis (diseases of the liver, but especially hypertrophic biliary cirrhosis), (4) unilateral

clubbing, with aneurysm of the aorta or of the large branches thereof, with pressure upon the principal nerves to the affected extremity. Locke's own catalogue of the principal etiologic factors is as follows: "The most common causes arranged in the order of frequency are bronchiectasis, pulmonary tuberculosis, empyema, malignant disease of the lungs and mediastinum, valvular heart disease, congenital heart disease, biliary cirrhosis, chronic jaundice, and chronic enteritis."

Both Bamberger and Marie emphasized the relationship of pulmonary disease to secondary osteo-arthropathy. Collected statistics have justified their insistence upon the importance of pulmonary disease as a primary disease. In Thayer's series pulmonary disease was present in 43 out of 55 cases, in Janeway's, pulmonary disease was present in 65 of 93 cases, and in Wynn's, 61 out of 100 cases had associated pulmonary disease. Locke in 1915 collected 139 cases, to which he added 5 of his own, a total of 144. Some disease of the respiratory tract was the associated condition in 112 of these.

The incidence of hypertrophic osteo-arthropathy in the course of pulmonary tuberculosis has been a favorite subject for discussion since the first description of the disease. In fact, for many generations the association of clubbing of the fingers with tuberculosis had given rise to many expressions of opinion as to the diagnostic and prognostic significance of the changes in the fingers. Though these views as to the diagnostic value of the clubbed fingers have only a historic interest now, the frequent occurrence of clubbing with pulmonary tuberculosis is a matter of fact. The interest in the facts has shifted from the diagnostic value of clubbed fingers in tuberculosis to the theoretic considerations of the relationship which the primary disease bears to the changes in the bones and soft parts manifested in secondary osteo-arthropathy. Some twenty years ago a report was published from the Brompton Hospital covering 600 cases of pulmonary tuberculosis; 69.6 per cent. of the male patients and 66.4 per cent. of the female patients showed some degree of clubbing. For many years the opinion prevailed that clubbing is common in tuberculosis, but the changes in the bones found

in the more advanced stages of secondary osteo-arthropathy are rare. That this view is probably incorrect was shown by the studies of Kessel, who investigated about 100 cases of tuberculosis radiographically, and found (1) 5 patients presented simple well-defined clubbing of the fingers without bone changes, (2) 17 of the patients showed clubbing of the fingers with bone changes in the phalanges, (3) 10 patients showed clubbed fingers and changes in the long bones. These figures indicate an incidence of changes in the long bones in 10 per cent. of the cases of pulmonary tuberculosis taken as they come. It has been rather widely believed that the incidence of the osteo-arthropathy has been greater in those cases of tuberculosis accompanied with cavitation and secondary infection; this view may have to be materially modified. It is possible that changes in the bones are present early in many cases, but accessible to clinical demonstration only in the more advanced cases. The rôle of cavitation with secondary infection may prove to be less important. These facts are of significance, as will appear more clearly in connection with the discussion of pathogenesis; it makes some difference in our final ideas regarding the exciting cause whether these secondary bone changes oocur only after the development of secondary infection with extensive destruction of lung tissue and consequent interference with respiration, or whether they may and do occur with comparatively minor anatomic change and practically a simple tuberculous infection.

It is easy enough to name the diseases with which osteo-arthropathy is most frequently found; to tell you the "why" of this association is "something else again." Two principal hypotheses have been proposed, were proposed, indeed, by Bamberger, and have been the subject of almost endless discussion since. Is the osteo-arthropathy the result of a toxin or of a disturbed circulatory condition? In his first communication he puts the question as to whether hypertrophy of the bones can result from passive hyperemia, and states that such overgrowth has been proved for inflammatory hyperemia. Later he attempted to determine the relationship of a toxin to the development of the bony changes by rectal injection of young rabbits

with the contents of bronchiectatic cavities. This particular experiment was without result. We are still entirely at sea as to the nature of the exciting cause and as to its manner of action. We are dealing with a disease which appears to occur as the result of two causes—sepsis and passive congestion. The sepsis, however, is most often located in the lung, which fact justifies a qualification of the idea that osteo-arthropathy is the result of sepsis alone; indeed, it carries an implication that sepsis alone is not an adequate cause for only that form of sepsis which is accompanied with respiratory disease is commonly followed by these bony changes. May either cause act alone? What determines the incidence of the disease? If the disease is one, how account for the apparent predominance of "clubbing," with circulatory disturbances and the great rarity of the late stages of the disease apart from some form of chronic sepsis? If sepsis is a necessary factor in the production of osteo-arthropathy, how may we account for the undoubted occurrence of unilateral clubbing, of which several cases have been reported, including 2 by Osler, and of unilateral changes in the long bones, of which a case was reported by Berent, as stated by Shaw? How may we explain the frequent association with intrathoracic tumors? These and other questions confront one who attempts to demonstrate the unity of the etiology. The literature is filled with speculative discussions and with widely variant expressions of opinion. As one reads over and over the reasons for accepting the toxic or circulatory hypothesis, or the endeavors to combine the two, the wisdom of Bamberger's words quoted from his first description and already referred to in this discussion is impressive, "the etiology of which in most cases is uncertain."

Clubbing of the fingers is more apt to exist alone or with minor degrees of bony change in cases of heart disease with peripheral stagnation, without demonstrable signs of pulmonary infection. Vascular turgescence is a principal anatomic cause of clubbing. The facts indicate that cyanosis with its associated venous stagnation is an essential causative factor to which the factor of sepsis adds a major share in the production of the bony changes. The significance of venous stagnation as an

effective cause of osteo-arthropathy was questioned by Bamberger, who sought to show that heart disease is so often accompanied by pulmonary infection that the circulatory disturbance need not be regarded as an etiologic factor. Högler's article, of quite recent date, contains practically the same assumption. Högler does not regard heart disease as an essential cause of "akropachie," the name he uses for the condition under consideration. He endeavors to show that the cases attributed to passive congestion can almost always be demonstrated to have some form of pulmonary disease, and remarks that "passive congestion plays no essential rôle and does not need to be extensively discussed today," adding that cases of high-grade akropachie occur in which passive congestion in the greater and lesser circulation is entirely excluded. We are not prepared to admit that the relationship of venous stagnation to hypertrophic osteo-arthropathy can be so easily explained away. What is the nature of this assumed infection in cases of congenital and valvular heart disease which presents no symptoms save those which are adapted to the demonstration of the septic theory of the causation of these bony changes? We have no valid reason for the assumption that the usual case of congenital heart disease harbors a focus of infection. That the venous congestion has a share, and an important share, in the causation of the changes in the extremities seems to us well founded. The occurrence of unilateral clubbing is difficult to explain upon the hypothesis that the circulatory stagnation is not a factor in its production. Whence the sepsis in these cases? Högler assumes pressure upon the brachial plexus, but of 7 cases of aneurysm cited by him as illustrative of unilateral clubbing, only one had definite signs of such pressure as he reports the histories. An eighth case, with an old dislocation of the shoulder, had neuralgic pains.

The confusion which exists in regard to the pathogenesis is well stated by Forschbach: "The occurrence of clubbed fingers in many purulent processes not involving the respiratory organs raises a question as to whether the clubbing is related to imperfect respiration. One is inclined in such cases to put the blame upon bacterial irritative products which pass from the foci of

suppuration or inflammation into the circulation. Since, on the other hand, clubbed fingers have also been observed with severe impairment of the gas exchange (severe congenital heart disease, *i. e.*, pulmonary stenosis), it cannot be denied that metabolic processes developed with permanently impaired gas exchange can produce trophic irritations in the sense of increased growth of the soft parts." Locke closes his discussion of this phase of the subject with a statement which is at once terse and complete: "At least two principal causes of hypertrophic osteo-arthropathy must be granted, namely, a toxemia, bacterial or otherwise, and stasis in the peripheral circulation. Either may induce the characteristic changes, the former most commonly, or both may act together." I cannot refrain in this connection from quoting the closing words of Bamberger's second contribution upon this subject. It pictures so well how little progress we have made through these years in really reaching the underlying facts of this clinical condition. "As it appears from the foregoing, I have not been successful in finding an explanation which is satisfactory, without qualification, for the osseous changes which appear not to be very uncommon in pulmonary and cardiac diseases. The same difficulties are in the way of such an explanation as exist for a single idea of clubbed fingers. Perhaps for the present it may be regarded as most probable that different causes can give rise to the processes occurring frequently in one and the same individual."

I have asked your attention to this rather lengthy discussion of an unusual disease for several reasons: partly that you might have the opportunity of seeing a well-marked case of pulmonary osteo-arthropathy and be prepared to recognize it again; partly that you might learn the diagnostic significance of these changes, that the condition is not a disease in its own right, but is a part of some primary disease, that the presence of these bony changes should always lead you to look for the cause, and partly to impress upon you one more gap in our knowledge; we really know very little about the real cause of this disease. Perhaps Braun's suggestion, that it is endocrine in origin, may prove to be more than hypothesis. Certainly the theories advanced thus far can-

not be accepted as proved. I have not discussed with you the hypotheses of Thorburn, who believed this to be a tuberculous condition, and of Godlee, who regarded it as syphilitic. These hypotheses have met with no support. A question which has baffled some of the best minds of the profession for more than thirty years ought still to be filled with interest for us. Especially is this true when the question is bound up with problems of internal respiration. We stand now where Ebstein stood some twenty years ago. "As Bamberger and, recently, Groedel have expressed themselves, manifestly many circumstances share in the development of this deformity; a unified conception is not possible at present, and we may well agree with the statement of S. West, 'Clubbing' is one of those phenomena with which we are all so familiar that we appear to know more about it than we really do."

We need spend but little time in a consideration of the differential diagnosis of this form of osteo-arthropathy. It can hardly be confused with any other type of disease affecting the bones or the joints with the possible exception of acromegaly. The characteristic changes in the terminal phalanges and the secondary character of the disease (its constant association with a primary condition characterized by sepsis or passive congestion) differentiate it almost at once from other forms of arthritis and chronic diseases of the bones. Some confusion has existed in the differentiation since Marie's first paper, in which one of the cases described was probably a case of acromegaly.

The diagnosis is much simpler with our improved use of the *x*-ray. Acromegaly is a disease in which the bones are enlarged, but the typical periosteal proliferations of secondary osteo-arthropathy are absent. The enlargement of the lower jaw is characteristic of acromegaly. We have admitted some question about the presence of joint changes in osteo-arthropathy. Joint changes are not a part of acromegaly. The presence of a primary disease is of great importance. Pain does not occur in acromegaly, though it may be present in osteo-arthropathy just as this patient has described. Glycosuria and evidences of pituitary tumor are, of course, lacking in osteo-arthropathy.

What is the primary disease in this case? The patient says he has had tuberculosis for five years, and evidently this diagnosis is the one which several physicians have made. There are, nevertheless, certain features in the history and physical examination which lead us to question the existence of pulmonary tuberculosis as the original disease. Three examinations since his admission to our service have been negative for tubercle bacilli. As a factor in the diagnosis I admit that the failure thus far to find tubercle bacilli is trivial. It is a rule in the hospital that we may not transfer to the tuberculosis wards suspected cases of that disease, but must first demonstrate a "positive sputum," and we have learned that many times a case which is apparently well developed will not show this "positive sputum" for a week or more; then a specimen may show great numbers of the bacilli. There are other facts about this boy which are more suggestive, I was about to say, convincing. Rather, they are calculated to inculcate skepticism as to the actual existence of pulmonary tuberculosis during these five years. Even the demonstration of the tubercle bacillus now would not be final proof; the patient has been in several "fresh-air hospitals," and has thus been put in a position to acquire tuberculosis if it did not already exist. It may be well to emphasize here that while it is important to "suspect" tuberculosis early, and to care for the patient and members of his family accordingly, it is not to be forgotten that not every case of chronic pulmonary infection is tuberculous; such cases are not lightly to be placed in institutions where they are certain to be continually exposed to tuberculosis.

This patient has not the physical findings which we expect in connection with a tuberculosis of five years' standing. Auscultation reveals diffuse dry and moist râles, the signs of a bilateral bronchitis, but not the fine râles or the changes in the voice and breath sounds which accompany tuberculous infiltration of the lung with consequent fibrosis if the patient is to survive such a process for five years. We find hyperresonance, not dulness, significant of emphysema, not of pulmonary fibrosis or of pleural thickening. The pulmonary disease, whatever

it is, has lasted for five years in a patient who is now only thirteen years old; at a time of life when the chest is developing and the bony thorax is soft and yielding and easily deformed by chronic disease, this chest does not show the results of fibrotic contraction of the lung, but does show an enlargement; the chest is barrel shaped, not phthisical, in appearance. Let us consult

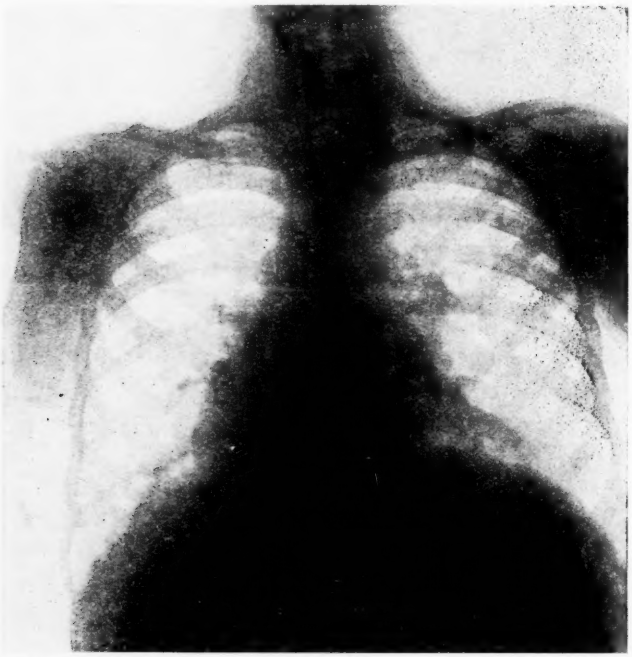


Fig. 130.—Radiograph of chest, showing changes noted in the text. Evidences of pathology about the inner and lower portions of both lungs.

the results of the roentgenologic examination. Here is the plate, and the report reads, "Hilum shadows dense with much indefinite peribronchial infiltration extending well through intermediate regions, being excessive adjacent to cardiac borders and causing irregular obscurity of cardiodiaphragmatic angles and rendering indefinite the outline of inner one-half to two-thirds of

each side of diaphragm. A glandular mass in right hilum may well contain calcification or caseation. Apices are comparatively clear. The fact that increased markings are greater in inner zone, and in this zone increase from upper to diaphragmatic regions render possible that the process is one of bronchiectasis with much passive congestion." In short, there is seen a lower lobe process with the apices clear, the process, roentgenologically, is not certainly tuberculosis.

While the significance of lower lobe involvement in excluding tuberculosis is of less importance in children than in adults, the *x*-ray adds no evidence for tuberculosis. In view of all the findings, clinical and roentgenologic, our opinion is that this is a case of bronchiectasis and not one of tuberculosis. The laboratory work demands some consideration, for the leukopenia and the anemia might well be interpreted as contradicting the opinion just expressed. It is not uncommon in long-standing septic conditions to find an absence of leukocytosis especially at a time when the patient is afebrile, as is the case here. The anemia may as well be a part of a non-specific infection as of one due to the tubercle bacillus.

As to some of the other symptoms of which the patient complained when he came to the hospital we are unable to offer any explanation. The abdominal pain of which he spoke has been slight since he came under our observation, and the diarrhea ceased almost immediately upon his admission. As to the significance of these symptoms we are at a loss for an explanation.

The prognosis of the osteo-arthropathy is dependent entirely upon the course of the primary disease. Since the patient has been in the hospital his general condition has steadily improved, and hand in hand with this improvement the swelling of the joints and of the extremities along with the pain of which he formerly complained so much has diminished. Until the primary disease is well the osteo-arthropathy will persist, doubtless will progress, but with remissions of the primary disease the patient is likely to have temporary relief from the symptoms associated with the disease of the extremities.

BIBLIOGRAPHY

V. Bamberger: Wien. klin. Woch., 1889, vol. ii, p. 226; Zeit. f. Klin. Med., 1891, vol. xviii, p. 193.
Hogler: Wien. Arch. f. Klin. Med., vol. i, p. 35.
Locke: Oxford Med., vol. iv, p. 431.
Ebstein: Deut. Arch. f. Klin. Med., vol. lxxxix, p. 87.
Kessel: Arch. of Internal Med., vol. xix, p. 239.
Kauffmann: Lehrb. der Path. Anat., vol. ii, p. 696.
Teleky: Wien. Klin. Woch., vol. x, p. 143.
Shaw: Allbutt's System, vol. iii, p. 64.
Forschbach: Schlayer and Ludke, Lehr. der Path. Phys., p. 529.

CLINIC OF DR. ARTHUR F. BYFIELD

COOK COUNTY HOSPITAL

THE DIAGNOSIS OF SPLENOMEGALY WITH HEMATEMESIS

THE three outstanding features of the case to be considered are: (1) The age of the patient, which is six years; (2) repeated hematemesis, and (3) an enlargement of the spleen.

We shall approach the diagnosis of this case as we frequently do, namely, by selecting one principal and undisputed symptom, reaching our conclusions from the evaluation of this symptom in its relationship to the clinical picture as a whole.

In the case before us we have a choice of two symptoms—hemorrhage from the upper portion of the gastro-intestinal tract and enlargement of the spleen—upon which to build a diagnosis; however, the more interesting phases of the splenomegalic picture have induced us to choose this as our starting-point. It may be noted, parenthetically, that the history of the case, the character of the bleeding, the features of the progress chart, the other points in the routine examination, and the outcome of the case all pointed unmistakably to hematemesis as against hemoptysis.

The history of the case can be briefly stated: This boy of six had seemingly been in perfect health prior to the onset of the present illness. He had never been of rosy complexion, it is true, yet his habitual olive tint resembled very closely that of his mother who is in good health.

Two days before he was brought to the hospital he had complained of pain in the upper abdomen unassociated with nausea or vomiting. In the late hours of the same day he had begun to vomit blood, this continuing at intervals during the following day. A physician who had taken care of the boy since infancy prescribed an expectant form of treatment, but this proving

unavailing the patient was brought to the hospital. It was estimated that he had vomited about 750 c.c. of blood during the two days prior to admission.

The remainder of the anamnesis is distinguished only by its negativeness: the boy had had no previous illness; the family history was quite negative, and the day's routine as to sleep, bowels, and appetite was entirely normal.

The initial examination showed the following:

1. Marked external evidences of hemorrhage, *i. e.*, pale skin and mucous membranes, dilated pupils, rapid low tension pulse, and restlessness. The respiration was not characteristic.

2. Negative lymphoglandular system, except for the spleen which is discussed separately.

3. Pleuræ and lungs were negative.

4. The heart was apparently within normal percussion limits and was otherwise normal except for a systolic blow over the base.

5. The abdomen was slightly distended, but definitely not rigid. There was moderate tenderness on deep pressure in the epigastrium. The liver was not enlarged either by palpation or percussion. (See Medical Clinics of North America, July, 1921, p. 152.)

6. The spleen was large and firm, with a rather sharp edge. It extended 3 to 4 fingerbreadths below the costal arch, moved freely with respiration, and was not tender.

7. On admission the temperature was normal, although the day following it was as high as 101.4° F. and remained at a variably elevated level throughout the patient's hospital stay. (The significance of the temperature curve will be considered in another place.)

8. The urine at the outset was normal in all particulars. Later a trace of albumin was frequently present and also, at times, acetone.

9. The benzidin test for blood in the stool was strongly positive in the first and many subsequent examinations, remaining so until about two weeks before the patient was discharged from the hospital.

10. The hematologic examination made on entrance read: Hemoglobin, 45–50; red blood-cells, 2,880,000; white blood-cells, 27,300 (first count) and 29,300 (second count). The differential count showed lymphocytes, 18 per cent.; monocytes (large mononuclears and transitionals), 5 per cent.; polynuclear neutrophils, 76 per cent.; polynuclear eosinophils, 1 per cent.

Summarizing briefly the data just given, we had to do with a child of six who, without warning, vomited nearly a quart of blood in a period of less than forty-eight hours. The examination showed, as might be expected, well-marked evidences of a recent severe hemorrhage and this was confirmed by the reduced number of red blood-corpuscles and the low hemoglobin. The marked leukocytosis was probably of the posthemorrhagic type. A slight epigastric tenderness, with some pain in the same region preceding the hematemesis, was perhaps of importance. Lastly and most significant because of its unequivocal character was a large firm spleen.

Upon the basis of the splenomegaly, therefore, let us attempt a critical study of the case.

Differential Diagnosis.—The following conditions deserved consideration in the differential diagnosis:

1. The spleen of hemorrhage.
2. The leukemias—acute and chronic.
3. Splenic anemia.
4. Malaria.
5. Cirrhoses of the liver.
6. von Jaksch's anemia.
7. Pernicious anemia.
8. Hodgkin's disease.
9. The purpuric conditions associated with bleeding from mucous membranes, *e. g.*, morbus maculosus (Werlhof's disease).
10. Hemolytic icterus.
11. Septic states.
12. Tuberculosis.
13. Syphilis.

Several of the rarer forms of splenic enlargement have purposely been omitted for the sake of brevity; and certain of the conditions mentioned overlap to some extent, but this fault can be remedied in the details of the differential diagnosis to which we shall now proceed.

1. *The Spleen of Hemorrhage.*—The possibility of this solution was prominently in our minds at all times until the diagnosis was finally established. In infancy and childhood the hematopoietic organs and the blood itself are in a state of uncertain equilibrium; immature and distinctly pathologic cells frequently appear in the blood-stream and denote no diseased state; the spleen is prone to enlarge upon slight provocation. Conditions are distinctly different from those of adult life and must be interpreted cautiously. The splenic enlargement in our case, therefore, might well have been the consequence of the severe loss of blood. The high white cell count (27,300 and 29,300) seemed to argue also for a brisk reaction to hemorrhage.

Speaking against this interpretation of the splenic tumor were, first, the size of the organ, for even in the quick responses of infancy and childhood to spleen-affecting states an organ extending nearly a handbreadth below the costal arch must be exceptional. In the second place, the firmness of the organ spoke against the spleen of hemorrhage; again, a definite increase in the size of the spleen, even at times when the blood was on the upturn, suggested other pathology. Finally, the later drop in the leukocyte count to normal and subnormal levels argued against the theory of hemorrhage.

At this point, before proceeding with our differential diagnosis, it seems logical to discuss what may have been the source of the bleeding if the splenic enlargement were regarded as hemorrhagic in origin. Naturally, we at once thought of peptic ulcer because of the initial epigastric pain and the later sensitiveness in this region. A history of preceding digestive disturbances was entirely lacking, yet we know that a severe, even fatal, hemorrhage may clinically usher in an ulcer. Again, while peptic ulcer is not common in childhood, it is by no means a rarity, and from autopsy records it would seem to be a lesion often over-

looked. We are familiar with the fact that in adults duodenal ulcer is far more common than gastric, and that in the former melena is more likely than hematemesis. Data are too meager to permit us to say that there exists in childhood the same ratio between duodenal and gastric ulcers or that a hemorrhage from the mouth argued somewhat against the (presumptively) more common, *i. e.*, duodenal, lesion.

The only other possible cause of gastric bleeding which was seriously considered, first assuming the splenic enlargement to have been secondary to such bleeding, was that associated with morbus maculosus, and this condition will be discussed presently.

2. *The Leukemias, Acute and Chronic.*—One might easily be tempted to say that the spleen could not possibly have been leukemic in origin because the hematologic data were so definitely non-leukemic. This ground must be carefully trodden, however, in view of what has already been said in relation to the instability of the child's hematopoietic organs. There is a group of childhood "blood" conditions which for the present cannot be classified; they represent pathology very distinctly and yet they are not readily grouped with the well-demarcated adult conditions. This is a very large and very obscure subject to which we cannot devote further attention now. Suffice it to say that the diagnosis of an atypical leukemia in childhood as well as in adult life is always difficult and sometimes impossible.

However, in the case before us, the several leukemias could not be seriously considered for the following reasons, even assuming that the leukocyte count of 27,000 to 29,000 could mean only a subleukemic state:

(*a*) In chronic myeloid leukemia the spleen is generally much larger, myelocytes dominate the blood-picture, and an acute onset with hemorrhage is unlikely.

(*b*) In the chronic lymphoid form the lymph-node enlargement is prominent and a very large percentage of lymphocytes is essential to a diagnosis.

(*c*) In the acute forms of either type a total white cell count as low as 29,000 is not uncommon at the outset and hemorrhages from mucous surfaces are frequent; yet these conditions must

be ruled out because of the absence of those diagnostic large immature cells (lymphoblasts or myeloblasts, as the case may be). As we have already stated, no pathologic white cells were present at any time in our patient's blood.

3. *Splenic Anemia.*—The onset with hematemesis of this initial stage of Banti's disease is well known. Most cases are first called gastric or duodenal ulcer and it is only the further course of events that permits of a diagnosis.

Our patient presented an enlarged spleen, plus anemia, plus initial gastric hemorrhage. But we were next confronted with one great objection to this diagnosis, namely, the high leukocyte count with 76 per cent. polynuclear neutrophils instead of a characteristic leukopenia, with a relative lymphocytosis. The age of the patient was not a determining factor because cases of splenic anemia are occasionally encountered in childhood.

The diagnosis of splenic anemia could still be acceptable if one called the high white cell count a posthemorrhagic leukocytosis.

4. *Malaria* was hardly a serious diagnosis; even if other things were confirmatory, the hematemesis would definitely eliminate such a possibility.

5. *Cirrhoses of the Liver.*—These conditions were included in the differential diagnosis because of the splenomegaly and hematemesis. The case could hardly be considered a cirrhosis of the liver in the absence of any demonstrable liver change. Diagnosis of cirrhosis upon a splenic change alone practically identifies the cirrhoses with Banti's disease, a hypothesis which has much in its favor. (See Medical Clinics of North America, May, 1922, p. 1587.)

6. *von Jaksch's Anemia Pseudoleukemica Infantum.*—One of the most characteristic features of this condition is its blood-picture which is rich in pathologic cells of nearly all types—nucleated red cells, myelocytes, immature lymphoid cells. This variegated picture was entirely lacking in the blood of the case we are considering.

7. *Pernicous anemia* is occasionally observed at this early age. We have always held to the strict interpretation of per-

nicious anemia which calls for a high color index, large-celled type of anemia, leukopenia, etc. In this sense our case, with a color index generally less than one, with red corpuscles which tended largely to small types with pale centers, and a white cell count more often higher than normal rather than lower could hardly be called a pernicious anemia. Furthermore, a large spleen is the exception in pernicious anemia and an initial gastric hemorrhage a rare thing.

8. *Hodgkin's disease* may for a variable period run its course with splenomegaly alone (splenic type). Hematemesis, however, made this diagnosis highly improbable.

9. *Morbus Maculosus of Werlhof.*—This is a type of hemorrhagic disease occurring particularly in young, delicate individuals, especially girls, and characterized by purpura plus bleeding from mucous surfaces. It is certainly not a disease entity, some forms being secondary to other conditions (acute leukemia, sepsis, etc.). A large spleen is not considered characteristic of the idiopathic form; furthermore, in our case there were at no time purpuric spots.

10. *Hemolytic Icterus.*—This has only the enlarged spleen in common with our case. There was at no time jaundice, and with hypotonic salt solutions hemolysis began at 0.45 and was complete at 0.36.

11. *Sepsis.*—Septic conditions are often associated with a splenic tumor and with a tendency to hemorrhage. The febrile course of our case was also suggestive of a septic state. Against a septicemia of the usual type was a persistently negative blood-culture, a constantly normal endocardium, the absence of joint symptoms, etc. Repeated large hemorrhages are also not common in the usual septic picture.

12, 13. *Tuberculosis, Syphilis.*—We believe that tuberculosis and syphilis could be considered as possible factors in the splenic enlargement only in so far as they might play a part in producing the splenic stage of Banti's disease. Pathologists generally are agreed that syphilis and tuberculosis are etiologic factors in certain of the symptomatic Banti cases. If, therefore,

our case was to be regarded as a splenic anemia, the discussion of the etiology must include lues and tuberculosis.

We have now considered in some detail the more important causes of splenomegaly in relationship to the particular case in hand, and we think it justifiable to limit materially the diagnostic possibilities. The types of splenomegaly just considered which remained for further consideration were:

1. The spleen of hemorrhage, the latter being the result of a bleeding gastric or duodenal ulcer.

2. Splenic anemia, either in the idiopathic (Banti) sense or as the result of some toxic or infectious process (lues, tuberculosis, or sepsis).

A brief review at this point of the progress of the case will throw considerable light upon our further efforts to come to a diagnostic conclusion, as between the two conditions just mentioned as probabilities. Despite the seriousness of the case the course was altogether a rather monotonous one, *i. e.*, for about three weeks the boy's condition did not change greatly from day to day. The outstanding events of these first three weeks were:

1. A continuous fever of the septic type, the peak ranging from 101° to 103° F., the pulse-rate being proportionately accelerated.

2. Five transfusions of citrated blood, the first shortly after admission to the hospital; the second, the following day, after a fresh hemorrhage; the third, at a point where the red corpuscles reached their lowest level (1,500,000); the fourth, following another fresh hemorrhage which occurred nearly three weeks after admission; and the last, at the time of operation.

3. The red blood-count remained at 2,000,000 or slightly above, with the exception of two days, during the entire hospital course of the case. The white blood-count pursued a course difficult to understand at the time, but fairly clear now that the case has been classified. The high counts on admission (27,300 and 29,000) had dropped to 12,000 on the third day. Thereafter, for two weeks, there was a distinct leukopenia (as low as 2800), generally without relative lymphocytosis, with the exception of two days when the white cells numbered 7100 and 7300. At

no time was there a true relative lymphocytosis, bearing in mind the patient's age. Following operation, performed three weeks after admission, there was first a slight leukocytosis succeeded by a practically normal count.

Two facts in particular tended to strengthen us in the opinion we had formed after our first study of the case, namely, that the condition was one of splenic anemia. These were the gradual appearance of a leukopenia, the count falling as low as 2800, and a slight but progressive enlargement of the spleen. Despite our feeling that splenectomy offered the boy his only hope, we permitted ourselves to be swayed by the wishes of the parents, who felt that so long as there existed a remote possibility of ulcer they preferred ulcer management to operation. The ulcer therapy consisted of initial small milk and cream feedings, with heavy alkalinization, followed by purées and soft diet.

As the boy's condition showed little improvement under this régime, further transfusion, indeed, being called for by a fall in the red corpuscles to 1,500,000, and the stools persistently showing a strongly positive benzidin reaction, operation was again urged. At this point the parents called for a gastro-intestinal *x*-ray examination which until then had not been undertaken because of the recent bleedings. The examination made by a competent roentgenologist under necessarily difficult conditions showed inconclusive evidence of duodenal ulcer. Such duodenal deformity as was visualized was not constant.

Two days later there was a fresh hemorrhage requiring another transfusion, and the following day the parents consented to a laparotomy. This was done by Drs. Richter and Buchbinder and consisted of exploration, followed by splenectomy. The stomach and duodenum were found quite normal, the spleen showed no firm adhesions, and the liver was apparently normal.

The pathologist's report of the organ is as follows:

"*Gross Examination.*—The spleen as received measures 12.4 x 8.7 x 6.3 cm. and weighs 246 grams. It is pink in color, of firm consistency, and exhibits a capsule roughened in places by a thin, flaky deposit which may be scraped away. The blood-vessels near the hilus are rather thick walled and contain a small amount

of light colored fluid blood. The organ is uniform in appearance and consistency.

"The spleen cuts with increased resistance. The cut surfaces are pinkish and moist and the reticulum stands out as fine, white intermingling threads. Scattered over the cut surfaces are numerous, minute, glistening dewdrop-like elevations.

"*Summary.*—The gross appearance of the organ is that of a chronic splenitis, associated with a fibrinous perisplenitis.

"*Microscopic Examination.*—Paraffin sections made from several areas of the organ show a marked increase of reticulum. Many areas resembling lymph-follicles are seen scattered throughout the section. The capsule is only slightly thickened. The fibrous trabecular structures are prominent and the blood-vessels are increased in thickness. The sinuses are greatly dilated and are loosely lined with very small round cells. Stained specimen shows no tubercle bacilli or Treponema pallida.

"*Diagnosis.*—Chronic splenitis of the Banti symptom-complex type."

The boy remained in the hospital for a month after the operation. During this time his condition showed improvement in some particulars and none in others. His appetite improved and his weight increased under the more liberal diet permitted. His appearance as to color was much better, and he became brighter and slept more quietly. There was no further hematemesis and the blood gradually disappeared from the stool. The hematologic condition, on the other hand, remained practically stationary and, as already noted, the fever persisted. Finally, just a month after operation, the boy's general condition appeared so satisfactory that he was permitted to go home despite his fever.

We have seen the patient only once since he left the hospital. At this time, about seven months after operation, he appeared to be a perfectly healthy boy and his general physical examination was negative. The red cells numbered 3,980,000, the hemoglobin 60, and the leukocytes 9550. In addition, there was possibly a slight relative lymphocytosis. More recently we have been informed that he continues to be well and that his blood is

quite normal. The fever subsided shortly after he left the hospital.

We feel that our diagnosis of splenic anemia in the case is correct, but we do not feel that we have thereby offered a complete diagnosis. Is this a case of idiopathic splenic anemia—Banti's disease in the strict sense—or is it a symptomatic form of the disease which in all likelihood is by far the commoner of the two forms? Was the long-continued fever an "anemic fever," which our experience must make us believe does exist, or was the fever a manifestation of an infectious or toxic process exerting its action primarily upon the spleen and producing a symptomatic splenic anemia? A negative Wassermann seemed to exclude syphilis as a cause, tuberculosis was unlikely in the light of histologic examination, and there was little to speak for a chronic septic condition. This, like many other splenic anemias, must remain hazy from the etiologic standpoint.

CLINIC OF DR. EDWARD LYMAN CORNELL

CHICAGO LYING-IN HOSPITAL

PYELITIS COMPLICATING PREGNANCY

I WISH to take up for discussion this morning the subject of pyelitis complicating pregnancy.

This patient, Mrs. M. B., No. 37,034, presents a rather interesting history. She is thirty-three years of age, para *i*. Her father died an accidental death and her mother died after childbirth, apparently from puerperal sepsis. She has 6 sisters and 1 brother living and well. She states that she has never been sick, denying the usual diseases of childhood.

Her menstrual periods began at fourteen years of age, twenty-eight-day type, lasting four to five days, with a moderate amount of flow and some pain. Her last regular period was October 27, 1923, and she felt the baby on March 5, 1924.

She had slight nausea in the early months of pregnancy, but no vomiting. At the time of the first examination in January she complained a little of constipation, but had no headache, edema, or bleeding. She stated that the September period was quite irregular in the amount of flow and time of appearance.

On examination her general condition was good. Her heart, lungs, teeth, throat, ears, and extremities were all negative. She was $61\frac{1}{2}$ inches tall and weighed 103 pounds. The pelvic measurements were 27 intercristal, $28\frac{1}{2}$ interspinal, bitrochanteric 31, Baudelocque 18, and conjugate diagonalis 12. The blood-pressure was 140/90/86. The blood-pressure reports and memoranda at subsequent examinations were as follows:

Date.	Blood-pressure.	Weight, pounds.	Condition.
1/30/24	110/70/66	106	Occasional headache, constipation. Alophen pills prescribed.
3/14/24	100/64/60	113	Sore throat for three weeks until now. Patient away visiting.
4/12/24	104/64/58	116	Feels fine.
6/5/24	Patient entered hospital.		

The urinalysis reports were as follows:

Date.	C.c. in twenty-four hours.	Specific gravity.	Reaction.	Albumin.	Sugar.	Bacteria.
1/9/24	3000	1010	Acid	Trace	0	+++
1/29/24	3000	1010	Acid	Trace	0	++
3/4/24	3000	1010	Acid	Trace	0	— —
3/25/24	2500	1011	Acid	Trace	0	+
5/8/24	2500	1010	Acid	Trace	0	+
5/25/24	2500	1016	Alk.	Trace	0	+

The latter part of May the patient took an automobile ride in a small car over rather rough country roads to a lake resort. On this road the machine went over a heavy bump, throwing the patient to the top of the car. The next day she complained of some pain in the back. The second day the pain in the back was much more severe and was accompanied by burning on urination. She was not sure that she had temperature, but she had a feeling of general lassitude. She noted that the pain in the back was more marked on the right side. On the third day she called a local physician, who stated that she had a temperature, the elevation not being mentioned. He put her to bed on a light diet and told her to drink plenty of fluids. She gradually became worse, the pain in the back becoming very severe, and the dysuria quite pronounced. She returned to Chicago June 5th and entered the Chicago Lying-in Hospital, complaining of severe, more or less constant, pain in the region of the right kidney, and burning on urination accompanied by considerable pain during the entire act.

Physical examination at this time revealed marked tenderness over the right kidney and along the right ureter and over the bladder. The chest and other portions of the abdomen were negative. Urinalysis made at this time showed a specific gravity of 1020, acid reaction, a trace of albumin, no sugar, and large amounts of pus. The white blood-count was 14,500.

On entrance the patient was put to bed and told to lie on the back or left side, and placed on a non-protein diet. Fluids were forced, especially fruit juices, such as lemon, orange, and grape-fruit. Urotropin, 10 grains three times a day by mouth, was prescribed.

The temperature on entrance was 99.6° F. It gradually receded until the fourth and fifth days, when it was normal.

Date.	Temperature: A. M.	P. M.	Pulse: A. M.	P. M.
1st day	....	99.6	..	108
2d day	97.6	99	84	92
3d day	98	98	80	100
4th day	97.4	97.8	80	80
5th day	98	98.4	72	84

Examination of the specimen of urine on June 7th showed practically the same findings as on the 6th. The examination on June 9th showed a specific gravity of 1014, acid reaction, small trace of albumin, occasional pus-cells. She was discharged from the hospital on June 10th, at which time she felt very comfortable except for an occasional pain in the right kidney area and a little dysuria. The blood-pressure readings and memoranda for the next few weeks were as follows:

Date.	Blood-pressure.	Weight, pounds.	Condition.
6/27/24	108/60/54	124	Complains of twitching in legs.
7/8/24	122/82/76	126	Feels poorly; complains of pain in back and on left side. Examination shows a tender left kidney. Urotropin, forced fluids, and non-protein diet prescribed.
7/18/24	110/72/66	127¾	Pain in back somewhat improved, but patient is unable to sleep. Allonal tablets prescribed in addition to the urotropin.

The urinalysis made during this period showed the following:

Date.	C.c. in twenty-four hours.	Specific gravity.	Reaction.	Albumin.	Sugar.	Microscopic.
6/27/24	3500	1010	Acid	No	No	Bacteria and urates
7/4/24	2750	1020	Acid	No	No	Negative
7/12/24	2500	1010	Acid	No	No	Negative
7/17/24	2500	1015	Acid	No	No	Negative
7/19/24	Single	1010	Acid	No	No	Negative

The patient entered the hospital in labor on July 25th at 3.13 A. M. The pains began at 10.30 A. M. on the 24th, and were

of moderate strength, the intervals varying from eight to fifteen minutes. Her blood-pressure on entrance was 108/60. She had no bloody discharge.

On external examination the ovoid was longitudinal, the head in the inlet, the breech in the fundus, the back on the left. The head was engaged and the diagnosis was O. L. P. She complained bitterly of the pain in the left kidney area. For the past few days this had been increasing in severity. On entrance her temperature was 98.2° F.; at 7 o'clock in the morning her temperature was 97.6° F., pulse 88, and respiration 26.

Rectal examination made at 4.30 in the morning showed the membranes intact, the cervix effaced, and 4 cm. dilatation. The top of the baby's head was on a level with the internal spines. The fetal heart tones were normal. She was having pains every eight minutes. At 5.45 A. M. a rectal examination showed the same findings except that the dilatation was 7 cm. The pains were at that time strong and appeared at five-minute intervals. The head was then in the transverse position.

During all this time the patient was complaining as much of the pain in the back and in the left kidney area as she was of the labor pains. At 7.30 A. M. the dilatation was complete. The bag of waters was intact. At 8 o'clock, the bag of waters not having ruptured, the patient was prepared for delivery. At 8.29 the bag of waters was artificially ruptured and the baby was born at 8.36 A. M. An episiotomy was done, and after delivery it was repaired, using catgut and silkworm-gut. The baby was a girl, weighing 3240 grams, and was in good condition. The patient was given $\frac{1}{2}$ c.c. of pituitrin at 8.36 A. M. and 1 c.c. of ergot at 8.54 after delivery of the placenta. She had an ether anesthesia.

At noon of the same day her temperature was 98.2° F., pulse 70. At 4 o'clock her temperature was 100.2° F., pulse 80. She was fairly comfortable at the latter hour, but at 9 o'clock in the evening she complained of pain in the small of the back, together with some gas-pains. Allanol, 2 tablets, was prescribed at 10.45 P. M., and the patient slept well during the night. On July 26th the morning temperature was 97.8° F., pulse 70.

The highest temperature occurred at 8 o'clock in the evening. She complained of pain in the small of the back and some dysuria. On the third day the pain in the back was very severe and 3 allanol tablets were prescribed. On the evening of the fourth day her temperature was 101° F., and the pain in the kidney area was so severe as to require a narcotic. The accompanying chart (Fig. 131) records the temperature.

Pyelitis usually appears in the last trimester of pregnancy rather than in the early months. There are certain features of

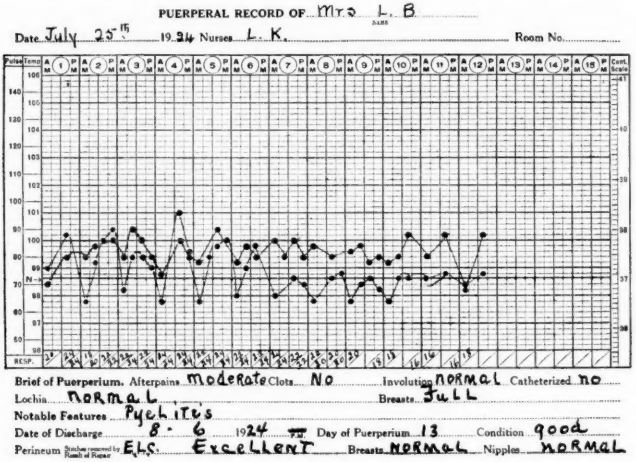


Fig. 131.

pyelitis in pregnancy that I wish to stress. We will take up first the diagnosis. These patients usually first complain of pain in the back over the kidney area. A little later it is accompanied by burning on urination. Occasionally the bladder symptoms appear first and the pain in the back second. There is frequency of urination and unless the patient is treated the condition rapidly becomes worse. The patient feels as if her bladder was never empty even after urination. The pain and burning is usually bitterly complained of and, all in all, these women are very uncomfortable. The temperature in these cases

is usually not high except where the ureters are blocked and there is a retention of urine and pus in the kidney pelvis. The bacterial content of the urine is usually of the colon type, though occasionally we find the staphylococcus and exceptionally the streptococcus. The urine is usually swarming with bacteria. The amount of pus present may be small or large.

In the very acute type of pyelitis there may be a chill or several chills, accompanied by pyemic fever. These patients occasionally vomit. All the other symptoms mentioned are usually greatly exaggerated. It is seldom that pyelitis is present without symptoms, but occasionally we find large quantities of pus in the urine, and still the patients complain very little.

The differential diagnosis presents considerable difficulty especially in many cases of right-sided pyelitis. Conditions occasionally simulating pyelitis are cholecystitis, cholelithiasis, renal stone, Dietl's crisis, appendicitis, ovarian cysts, tubal pregnancy, and salpingitis. I am not going to enter into a full discussion of each of these, but if a careful physical examination is made it is usually not so difficult to rule out these various conditions. A case of twisted ovarian cyst accompanied by pus in the urine gave me considerable difficulty in the diagnosis several years ago. This patient complained of constant pain in the lower abdomen with marked rigidity over the rectus muscle. She had a low white count, no temperature, and no pain over the kidney area. On ureteral catheterization there were no signs of blocking. She was about three months pregnant, and a tumor mass could be felt on bimanual examination. Chronic or acute appendicitis seldom accompanies pregnancy, while pyelitis is not an infrequent complication. The urinalysis and the location of the pain, together with the type of temperature, usually suffices to differentiate this condition.

Left-sided pyelitis usually presents little difficulty. During the flu epidemic of 1918–1920 I saw several pregnant women who were diagnosed as influenza patients by the general practitioner, the diagnosis being based on pain in the back, temperature, and headache. One patient in particular claimed to have had four attacks of flu from January to March. In the last

attack I was called in consultation, and we found a typical case of double pyelonephritis.

In making a diagnosis of pyelitis the cystoscope should be used on all doubtful cases. The ureteral openings are swollen and inflamed and pus is seen flowing from them. The bladder may or may not show signs of cystitis. On catheterizing the ureter, urine filled with pus is obtained. The pyelitis may or may not be unilateral. Most of the patients experience almost immediate relief of the pain as soon as the catheter reaches the pelvis of the kidney. This is especially noticeable in cases where the ureter is blocked.

Occasionally these patients show such a toxic condition that may simulate any of the toxemias of pregnancy. Just recently a patient was admitted to the hospital two weeks from term complaining of pain in the back, frequent urination, dizzy attacks, spots in front of the eyes, edema of the legs and eyelids, shortness of breath, and slight pains in the epigastric region. She had a temperature of 100° to 101° F., a pulse ranging from 80 to 90, and a blood-pressure averaging 140/80. Urinalysis showed a large quantity of pus. She came in with a diagnosis of threatened eclampsia. On careful physical examination the pyelitis was located on the right side, and on appropriate management the symptoms of eclampsia disappeared at the end of four days, the blood-pressure returning to normal. This patient's ureter on the right side was catheterized and the catheter left in for twenty-four hours. She was a primipara, and labor was induced by castor oil and quinin one week after her entrance to the hospital. The point that I wish to bring out is that pyelitis may be the etiologic factor in producing eclampsia or other forms of the toxemia of pregnancy.

Fortunately the management of the pyelitis of pregnancy usually presents very few difficulties. It is, strictly speaking, a medical situation. These patients should be immediately placed in bed, put on a bland diet, forcing the fluids, and given urinary antiseptics. I usually issue the following orders on entrance to the hospital:

"Patient continuously in bed; 4000 c.c. of fluid by mouth

for twenty-four hours; plenty of fruit juices; milk, buttermilk, toast, and crackers; hexamethylenamin, grains 10, four times a day, a twenty-four-hour urinary examination, complete blood-count, pulse, temperature, and respiration every four hours; hot-water bag to the kidney area."

If the dysuria is quite severe I prescribe hyoscin hydrobromid, grain 1/1000, four times a day. When the acute symptoms subside a bland diet is prescribed, and the hexamethylenamin decreased in amount. If the pain is very severe at the end of thirty-six hours the patient is cystoscoped and the ureteral catheter left in the affected side. Usually $\frac{1}{2}$ per cent. silver nitrate solution is put into the pelvis of the kidney. Lately I have been using one ampule (5 c.c.) of uritone intravenously, instead of hexamethylene by mouth.

The patient under discussion has been under this management except that we have not catheterized her. I did not think it advisable to catheterize her so soon after delivery unless the indications are more pronounced than they are now. This morning she received her first injection of uritone. I shall let you know at a later time the results of this form of medication.

I hardly feel that the interruption of pregnancy is indicated in these patients nowadays. Since the introduction of ureteral catheterization I have found that most of them get relief from the pyelitis almost immediately and that they continue to improve and carry to term. Very exceptionally is it advisable to interrupt pregnancy. This is done when the patient is at or near term.

This patient left the hospital on the sixteenth day. She had been up and about for four days. She had some slight pain in the left kidney area when she left the hospital. She was weak and she tired easily. The urine was clear of pus. The uritone was well tolerated. The baby was nursing well and gained in weight without supplemental feeding. Six weeks later she reported she was quite well. The pain in the back and dysuria have entirely disappeared.